NEURITIS AND NEURALGIA

WILFRED HARRIS

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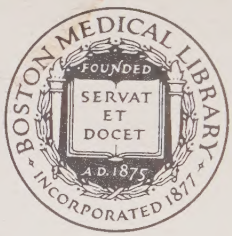

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OXFORD MEDICAL PUBLICATIONS

NEURITIS AND NEURALGIA

BY

WILFRED HARRIS

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THIS BOOK I DEDICATE

TO

MY WIFE

WHOSE ENCOURAGEMENT AND HELP HAVE BEEN

INVALUABLE IN ITS PREPARATION

PREFACE

THERE is no necessity to apologise for the appearance of this book. In it I have made some attempt at co-ordinating the knowledge gained by clinical researches in different types of Neuralgia, a disease of elusive pathology, whose classification and boundaries are therefore variable and elastic. In this volume I have given my own views, based on experience of over eight hundred cases of trigeminal neuralgia, and of numerous other and rarer forms. Incidentally the path of the taste fibres seemed to call for detailed analysis, for the views hitherto accepted do not appear to me to fit the facts.

If the pathogeny of what may be called the organic neuralgias is difficult, what shall be said of Migraine and its numerous family of migrainous neuralgias? The latter term I have employed to include some varieties of cranial neuralgia, mostly facial, whose superficial resemblance to migraine is not great, and which might be, and are, constantly mistaken for trigeminal neuralgia or other conditions.

The discussion of Neuralgia inevitably introduces that of Neuritis, for it is often difficult to separate one from the other. Like animal and vegetable life, which are practically indistinguishable in their unicellular forms, so do neuritis and neuralgia intermingle when morbid processes affect the finer nerve terminations. It seemed impossible to write of one without dealing fairly by the other; thus the title of the book was settled.

With such a subject some omissions are unavoidable, for which I crave indulgence, hoping that my results here

set out may help others to the understanding, and to further improvements in the treatment, of these refractory diseases.

WILFRED HARRIS.

56, *Wimpole Street,*
London, W.
January, 1926.

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NEURITIS AND NEURALGIA

CHAPTER I

INTRODUCTORY

The term neuritis means nerve inflammation, but the word is very loosely used, and now that it is a term familiar to the public at large it is applied to neurasthenia, acute delirium, facial neuralgia, sciatica and chronic pains of every kind. Indeed, knowing the soul-satisfying nature of the word, the family doctor may often camouflage the shooting pains of tabes to inquiring relatives by diagnosing "neuritis."

Active inflammatory lesions of the outer nerve sheaths, as well as parenchymatous degenerations of the axis cylinders and medullary sheaths, and chronic neuronic degenerations of the fibres, are all fairly included under the term neuritis, a large class which may be subdivided into multiple neuritis, which is always due to toxæmic circulatory causes, and local forms of neuritis, in which the outer fibrous sheaths of the nerve trunks, large or small, are damaged by pressure, local contiguous inflammation or bacterial invasion.

Pain in neuritis varies much in its intensity, and is most severe in lesions of the nerve sheaths, or perineuritis, in which not only are the nerve filaments supplying the sheaths, the nervi nervorum, themselves involved, but the inflammatory process, tending to spread inwards from the connective tissue outer sheaths, involves the intraneural bundles, compressing them and aggravating the pain, which, at first local, now spreads widely along the whole

territory of the nerve trunk. Examples of this form of neuritis are so-called rheumatic brachial neuritis and sciatica. Irritative phenomena are the early symptoms, and in the majority of cases the clinical signs do not progress beyond local tenderness, general hyperæsthesia and pain. In a minority of cases signs of nerve damage from compression will appear, as loss of deep reflexes, muscular wasting, anæsthesia and trophic phenomena of the skin and nails in the extremities.

In all such cases neuritic pains may be severe, and the term neuralgia should not be used.

Neuralgia, or nerve pain, is a term employed to describe shooting, radiating pains which spread far wider than the territory of the nerve filament whose disease initiates it. Thus an acute pulpitis causes at first an aching tooth, a purely local pain. If unrelieved, this is apt to set up shooting lancinating neuralgia along the whole of the mandibular or maxillary nerve, over the whole area of the trigeminal nerve on that side, and even down the neck and arm to the fingers. Such widespread pain is purely reflex, and may be accompanied by other reflex phenomena such as salivation, lachrymation, flushing and vasomotor œdema, and later trophic phenomena such as greyness of the hair.

Violent and widespread neuralgia may therefore often be a sensory reflex from an acute neuritis of a few nerve filaments. Indeed it has been questioned whether idiopathic neuralgia, that is to say, a neuralgia dependent on no recognisable pathological process, holds any place in medicine, and one is tempted to assert that all neuralgic attacks are reflexes from an irritative focus somewhere. However, this is at present too sweeping a generalisation, and nerve pains such as migrainous neuralgia and neurasthenic headaches, abdominal migraine and the large group of psychalgias have as yet no definite material basis for their origin. The varieties of migraine possibly are due to vasomotor disturbances, an initial arterial spasm and subsequent dilatation, and the migrainous facial and

cephalic neuralgias may perhaps be due to compression of afferent trigeminal meningeal nerve fibres.

The psychalgias, however—mental pains—are less easily explained. Their origin may sometimes be due to local suggestion, as in the coccygodynias following a slight blow; or they may succeed a period of worry, with no local injury whatever to determine the suggestion of local pain. The neuralgia, therefore, being a disordered mental perception, will not follow anatomical boundaries, and will be influenced differently by treatment. Often of very many years duration, their true relation to a disordered mental process may not be fully brought out until a searching inquiry into family history discloses a tale of nervous breakdowns, hysterical attacks, insanity and other neuropathic evidence in near relatives.

Recurrent or persistent pain is so often an evidence of progressive disease in some organ or tissue, such as cancer, ulceration, abscess or inflammatory mischief of some kind, that recognition of the different forms of neuritis and neuralgia, and their accurate localisation, is a matter of prime importance to the clinician. Yet how often is the diagnosis of "neuritis" made without even examining the part when a patient complains to the doctor of chronic pains around a shoulder or hip.

The chronic pains following herpes zoster in later life, and the intensely severe and lasting pain in some cases of causalgia, are instances of the possibility of an intensely painful local lesion so affecting the central receptive and perceptive nerve-cell areas in the spinal cord, thalamus and cortex, that the pain may persist unaltered even though the nerve trunks above the original damaged nerve structure are completely severed. With the causalgia from gunshot wounds of nerves may be compared the violent central pains in the upper limbs and trunk following shortly after gunshot wounds causing spinal concussion of the cervical cord. Holmes has shown how these two forms of neuralgia resemble each other in their violent exacerbation by the slightest touch of cotton wool or

vibration, movement of the limbs, or emotion. Clinically somewhat resembling psychalgias, yet in these cases definite slight lesions are found in nerve and spinal cord, such as swelling and œdema, with small hæmorrhages.

Central pains are common in chronic lesions of the lateral and posterior portions of the thalamus—the syndrome described by Dejerine and Roussy, in which the persistent pain, according to Head and Holmes, may be due, not to an irritant lesion of the central ascending paths, but to the removal of an inhibitory control which the cortex normally exerts on the subcortical centres concerned in the perception of these sensations.

Psychalgias may therefore be understood to be due to a functional inhibition of this control, and may be contrasted with hysterical anæsthesias, in which by a different mental process the cortical perception of tactile and painful and other impulses coming from lower centres has been inhibited. Such cortical inhibition in hysterical anæsthesias can always be broken down by a sufficient increase of the sensory stimulus, as by the use of a strong faradic battery, but no mechanical method has yet been devised for the cure of psychalgia, and we have to rely on suggestion and psychotherapy to supply the lacking mental stimulus to replace the inhibitory control.

The following classification of the causes of persistent pain in diseases of the nervous system is arranged in six classes, progressing from periphery to centre, or from nerve-ending to cerebral cortex.

Causes of Persistent or Recurrent Pain in Diseases affecting the Peripheral or Central Nervous System.

1. *Peripheral* due to inclusion of nerve-endings in scar.
   - (*a*) For example, neuro-fibrositis, traumatic or rheumatic; adiposis dolorosa.
   - (*b*) Due to septic or to rheumatic terminal trigeminal neuritis :
     - (i) Chronic paroxysmal trigeminal neuralgia (Fothergill's disease, tic douloureux).
     - (ii) Chronic neuralgia of upper or lower jaw.
   - (*c*) Geniculate neuralgia.
   - (*d*) Glossopharyngeal neuralgia.
   - (*e*) Superior laryngeal neuralgia.
2. *Disease involving Nerve Trunks.*
   - (*a*) Supra-orbital neuralgia, infra-orbital neuritis.
   - (*b*) Multiple neuritis.
   - (*c*) Brachial or sciatic perineuritis.
   - (*d*) Tumours or gummatous neuritis :
     - (i) Trigeminal.
     - (ii) Spinal, *e.g.* neurofibromatosis, malignant growths.
   - (*e*) Causalgia.
   - (*f*) Cervical or first rib pressure.
3. *Lesions of Posterior Root Ganglia or Posterior Roots.*
   - (*a*) Post-herpetic neuralgia : trigeminal and spinal.
   - (*b*) Tabetic neuralgia.
   - (*c*) Other root scleroses.
   - (*d*) Chronic meningitis.
4. *Central Sclerosis of Fillet or Thalamus.*
   - (*a*) Intra-medullary growths. Pontine and medullary thrombosis.
   - (*b*) Syringomyelia and syringobulbia.
   - (*c*) Thalamic softening.
5. *Neuroses :* Migraine and migrainous neuralgias.
6. *Psychalgias.*

CHAPTER II

MULTIPLE NEURITIS

Multiple Peripheral Neuritis

Polyneuritis or symmetrical degeneration of the peripheral nerves was first described by Duménil in 1864, and subsequently by Leyden, Eichhorst, Grainger Stewart, Ross, Bury and innumerable other writers. Previously to its recognition as a special disease of the nerves such cases had been mistaken for different forms of myelitis or ascending paralysis. In the majority of instances of symmetrical multiple neuritis the morbid process is not strictly inflammatory, but consists of a parenchymatous degeneration of the axis cylinders or nerve fibrils, either sensory or motor in function, or of both together. The site of the chief nerve damage in polyneuritis varies considerably in different types. In many cases the main nerve trunks, such as the external popliteal, ulnar and anterior crural, may show parenchymatous degeneration of the nerve fibres and also inflammatory interstitial changes. In some the changes may be limited almost entirely to motor fibres, and in others to cutaneous sensory branches; in some degenerative changes are most marked in the finer terminal branches, while occasionally, as in diphtheritic neuritis, marked degeneration may be found in the anterior root motor fibres close up to the nerve cells in the anterior horn.

Pathogeny

Attempts at generalisation from these facts have produced the theory that the lower motor neurone is affected as a whole in some forms, as lead neuritis, and the term

" neuronitis " has been applied to them. In lead paralysis both nerve cells and nerve fibres undoubtedly may be affected alone or together, cortical cells in saturnine encephalopathy, and spinal anterior horn cells in the subacute and chronic forms of lead poliomyelitis. Recovery is less likely when nerve cells are attacked, and then the paralysis shows itself in muscular groups according to spinal segments, such as the intrinsic musculature of the hand (1st dorsal), and the Erb group of deltoid, spinati and flexors of the elbow. The extensor paralysis of the wrist and fingers is not segmental, but a neuritis limited to the posterior interosseous nerve. Like the supinator longus, now known as the brachio-radialis, which escapes in lead palsy, the radial extensors of the wrist are supplied mainly from the fifth cervical nerve, and yet they suffer with the other extensors in the forearm which are mainly supplied from the seventh cervical, a fact which demonstrates the neuritic distribution of the paralysis of wrist-drop rather than a spinal segmental neuronic affection.

Doubtless many poisons have a selective action on certain parts of the nervous system in preference to others. Diphtheritic toxin is particularly liable to affect the ciliary muscle in the eye; absinthe is peculiar in its excitant action on the cerebral cortex; lead affects motor neurones only, and mainly those of the posterior interosseous nerve, yet this poison also shows affinity for nerve cells both in the cerebral cortex and in the anterior horns. Alcohol, arsenic and diabetes are specially liable to affect sensory fibres, either alone or together with motor nerves, and pain as a symptom in these forms of multiple neuritis is prominent as compared with other forms such as lead neuritis and many forms of toxic polyneuritis.

Occasionally in polyneuritis examination of the cerebro-spinal fluid after lumbar puncture has shown an excess of protein, the fluid perhaps coagulating spontaneously and being of a yellowish colour—the so-called Froin's syndrome. This is usually evidence of stagnation of the fluid in the lumbar sac, due to compression above, as in meningitis

or tumour, but there is no evidence of obstruction in autopsies on polyneuritis, and the fluid quickly becomes normal with recovery from the neuritis. The changes in the fluid may be due in part to ascending lymphatic currents towards the spinal cord from the inflamed nerves.[17]

Probably all forms of multiple neuritis, except the familial varieties, are toxic in origin, due to some circulating poison in the blood, and therefore to speak of *toxic polyneuritis* as a special variety of multiple neuritis may be misleading. It is, however, convenient to use this term toxic polyneuritis to include the numerous forms of symmetrical polyneuritis met with whose etiology is obscure.

The most important and commonest causes are chronic alcoholism, lead poisoning and diphtheria. Alcoholic neuritis is certainly very much rarer in this country since the introduction of restrictions on the consumption of alcohol imposed during the Great War, and the same applies to America since the advent of prohibition. Lead poisoning, too, is very much less frequent than formerly, owing to the general increase in knowledge of its dangers and the sanitary precautions taken. Diphtheritic neuritis has been stated to be more frequent since the introduction of Behring's anti-diphtheritic serum in 1894, and such increase may certainly be ascribed, not to any toxic action of the serum, but to the greatly diminished mortality from the disease, and the much larger number of possible subjects for diphtheritic neuritis.

Classification of Multiple Neuritis

Multiple neuritis is best classified in our present state of knowledge on an etiological basis, rather than on pathological distinctions or clinical differences, and five main groups may be separated :

(1) Those dependent on the absorption of poisons introduced into the body.

(2) Those due to auto-toxæmia, or poisons developed primarily within the body.
(3) Infective causes, of an organismal nature.
(4) Cachectic.
(5) Familial.

The first group may usefully be sub-divided into two groups, those due to metallic poisoning and those produced in poisoning by various carbon compounds.

Polyneuritis

(1) *External Poisons.* —(*a*) *Metals:* Lead, arsenic, antimony, mercury, copper, phosphorus.
(*b*) *Carbon compounds:* Alcohol, carbon bisulphide, carbon monoxide, dinitrobenzol, sulphonal, chloral, chloretone, aniline, tetrachlorethane.
(2) *Auto-toxæmic.*—Diabetes (pancreatic deficiency), beriberi (vitamin deficiency), puerperal, leukæmic, relapsing, slow chronic, hæmatoporphyrinuric (enterotoxin).
(3) *Infective.*—Diphtheria, typhoid, paratyphoid, scarlet fever, measles, small-pox, pneumonia, influenza, gonorrhœa, septicæmia, rheumatism, parotitis (with or without uveitis), malaria, acute febrile polyneuritis, syphilis,

tubercle { general multiple neuritis, symmetrical neuritis of the feet, acute local brachial neuritis.

(4) *Cachectic.*—Chronic anæmia, malignant disease, senility, syphilis, tubercle, hunger-œdema (? vitamin deficiency).
(5) *Familial.*—Charcot-Marie-Tooth muscular dystrophy, progressive hypertrophic interstitial neuritis of Dejerine.

Polyneuritis varies much in the severity of its symptoms, and is often slight in degree, pains being complained of in the arms and legs, with some paræsthesiæ of the extremities,

hands or feet, and perhaps some tenderness of nerve trunks. Such a slight degree of polyneuritis was formerly common in alcoholism, and may be met with as a sequel to almost any toxic cause of neuritis, without the development of any of the more definite symptoms of paralysis of the extensors, muscular wasting, anæsthesia and loss of the deep reflexes.

Alcoholic Neuritis may be acute or gradual in development. It is more commonly the result of many years excessive indulgence in alcohol, generally spirits, such as gin and whisky, though all forms of alcoholic drinks may contribute to its development. It is a matter of speculation how the alcohol produces the nervous lesions, and the argument has been put forward that an auto-toxæmia is set up by the chronic gastritis due to the effect of the alcohol upon the gastric mucous membrane, which in turn acts as an intoxicant upon the nervous elements. Although it is certainly true that cirrhosis of the liver may co-exist with alcoholic neuritis, its absence has often been remarked, and the rarity of alcoholic cirrhosis of the liver in asylum post-mortem work is well known, although chronic alcoholism is a factor of importance in the production of some insanities. It is not improbable that the neuritis is caused by the direct effect of the alcohol upon nerve tissue. Strong alcohol of 80 per cent. and upwards destroys living tissue by coagulating the albumin, a process known as neurolysis when employed in the treatment of neuralgias, and it is not difficult to understand that the persistent bathing of the tissues with blood containing alcohol above a minute percentage may after many months or years set up the inflammatory and degenerative changes in the nerves of multiple neuritis. It is said to be commoner in women, and it may develop acutely after a long bout of heavy drinking, commencing with pains in the limbs, often with fever, intense muscular tenderness and cutaneous hyperæsthesia, with delirium and insomnia. In this stage the knee jerks and other deep reflexes are usually increased, though these

disappear generally within two or three weeks; there are tingling and numbness in the feet and hands, and very soon there is loss of sensation to painful stimuli of the skin of the extremities, though deep muscular tenderness, such as may be evoked by squeezing the calves, may persist for months. Owing to the weakness of the dorsiflexors of the feet there is a tendency to foot-drop, and a characteristic high-stepping or "steppage" gait will be noticed. This peculiar gait is, however, even more marked in peroneal muscular atrophy. The cutaneous reflexes are usually abolished, though occasionally there is hyperæsthesia of the soles, with exaggerated flexor response.

Tenderness too of nerve trunks on pressure, such as the external popliteal or ulnar, may be noticeable. In severe forms foot-drop and wrist-drop develop, with rapid wasting of the anterior tibial group of muscles and the extensor muscles in the forearms. In addition to total paralysis of these groups, there is generally considerable weakness of the remaining trunk and limb muscles, so that the patient is quite unable to stand or to use the arms. Contractures of the hamstrings and gastrocnemii and long flexors of the toes are frequent, which if untreated will delay the recovery of the power of walking.

Paralysis of the diaphragm due to involvement of the phrenic nerves is sometimes present, and is apt to be of serious significance, and pneumonia not infrequently supervenes. Many cranial nerves may also be involved, such as facial paralysis on one or both sides, or paralysis of one or more ocular muscles, usually the external rectus. Total third nerve palsy is rarely if ever seen due to alcoholism alone, though reflex iridoplegia may occur in the absence of syphilis, and nystagmus too may be due to alcoholism only. Retro-bulbar neuritis of the optic nerves, causing central scotomata, is a not uncommon complication, with resultant pallor of the discs and optic atrophy. This tragedy is more especially liable to occur when wood alcohol has been used in the manufacture of drinks. Paralysis of the vocal cords has also been described due

to involvement of the vagus nerves, which may also give rise to a rapid pulse.

The sphincters are comparatively rarely involved, though occasionally either bladder or bowel paralysis may occur in alcoholic neuritis. Excessive sweating of the feet and hands is common, as is also œdema of the ankles. Other trophic phenomena are wasting of the finger pads, ridging and incurving of the nails, and sometimes glossy skin. In chronic recurrent polyneuritis, either alcoholic or diabetic, perforating ulcer of the foot may develop. In some cases motor paralysis and wasting are scarcely noticeable, the sensory phenomena predominating, with pains, anæsthesia of the peripheral parts of the limbs, and ataxic gait. Loss of the deep reflexes is generally present in these cases, which increases the resemblance of the syndrome to locomotor ataxy, and such cases are sometimes called alcoholic, diphtheritic or diabetic pseudo-tabes, according to their etiology. Though they resemble tabes in the pains, anæsthesia, ataxy and loss of deep reflexes, they may usually be differentiated from tabes by the absence of sphincter involvement or ocular troubles. However, as said above, diplopia, pallor of the discs, Argyll Robertson pupils and sphincter paralysis may sometimes occur in chronic alcoholism. When muscular paralysis is present the electrical reactions in the extensors of the feet and hands will show partial or complete reaction of degeneration. When this is partial the faradic reactions may be only diminished, the galvanic reactions showing an especially sluggish ACC.

A difficult point in differential diagnosis may arise in cases of syphilis which have received prolonged treatment with courses of intravenous arseno-benzol preparations and mercury in various forms. Loss of knee jerks and Achilles jerks may be the premonitory sign of the development of locomotor ataxy, but, on the other hand, this loss of deep reflexes may be the result of the treatment, an early sign of polyneuritis due to the prolonged use of arsenic and mercury. The point is of great importance in prognosis,

and yet may be impossible to decide with absolute certainty. The absence of shooting pains, analgesia of the legs and inner side of the fore-arms, or of sluggishness of pupil reaction to light, will be in favour of polyneuritis as the cause, but it will be sound policy to postpone definite diagnosis for twelve months, at the same time calling a halt in arsenic and mercurial medication. At the end of a year the position must be revised, the reflexes re-examined and the Wassermann blood and cerebro-spinal fluid tests repeated.

If the knee jerks have returned, polyneuritis due to the arsenic and mercury treatment may definitely be accepted as the cause. With continued loss of the deep reflexes, and especially with positive results from the cerebro-spinal fluid tests, the probability of latent or developing tabes is greatly increased.

Alcoholic neuritis is not infrequently associated with pulmonary tuberculosis, and a careful examination of the lungs should always be made in cases of polyneuritis. It is not improbable that here we have a combined action of the two toxins, alcoholism and tuberculosis, in the production of the polyneuritis. Arsenic and alcohol may act similarly in combination in producing polyneuritis, as in the well-known epidemic of polyneuritis in Manchester in 1899, where much of the beer had been contaminated with arsenic due to the glucose used in its manufacture having been made with an impure sulphuric acid derived from arsenical pyrites.

The heart is not infrequently diseased in cases of alcoholism, fibrosis, hypertrophy or sometimes extreme dilatation being common, and large liver too from hypertrophic cirrhosis may be present. Chronic renal disease and albuminuria may also occur with or without arteriosclerosis.

Polyneuritic Psychosis

The mental symptoms in alcoholic neuritis are often well marked. Loss of memory and unreliability of statement are common, and a history of sudden onset of

paralysis may be quite untruthfully given, while denials of alcoholic excess and even assertions of total abstinence must be critically weighed and not of necessity believed. A curious syndrome described by Korsakow in 1887 and known as *Korsakow's polyneuritic psychosis* is a well-known form of chronic delirium occurring in alcoholics, though it is by no means peculiar to alcoholism.

With loss of memory there is an extraordinary disorientation of space and time in patients who have been confined to bed for weeks; for example, they may assert that they were visiting distant countries on the previous day, with other similar wild and random statements. In several cases of polyneuritic psychosis of uncertain origin, though not alcoholic, I have seen permanent loss of memory and chronic mental failure, the man being quite unable to perform any but the simplest work. Several such cases occurred in soldiers who suffered from polyneuritis during the Great War, and the dependence of this peculiar loss of memory and permanent mental failure on a previous toxic polyneuritis appears not to be generally known. No doubt the toxæmia producing the psychosis has in such cases damaged the cells in the frontal cortex beyond repair, a toxic encephalitis.

Treatment.—As a rule total abstinence is essential, though exception must be made and the alcohol allowance reduced gradually when the drinking bout has been recent and excessive, or when the heart shows signs of failure. Complete rest in bed with plenty of sleep, artificially induced if necessary by five to ten grains of medinal, or bromidia in drachm doses. Occasionally morphine will be necessary to control the pains and to induce sleep, in combination with other hypnotics. The cutaneous tenderness and hyperæsthesia are sometimes so excessive that the limbs must be wrapped and bandaged with cotton wool. The feeding and nursing in acute cases are very important. Two-hourly feeds of milk, eggs and soups thickened with arrowroot or crême de riz, and later fish purées and soufflés, pounded meat and chicken broth.

When the pains and tenderness diminish light massage may be employed, followed later by galvanism. This may be given twice a day, the paralysed muscles being stroked with the anode, using a current of from seven to ten milliamperes. Injections of strychnine are also an invaluable remedy, one-fifteenth of a grain of the hydrochloride being given twice daily, this dose being continued for several weeks.

In order to obviate contracture of the gastrocnemii resulting from the foot-drop, the weight of the bed-clothes should be held off from the toes by rectangular padded splints applied underneath the calves and feet, the vertical foot-piece holding up the bed-clothes, and the foot and leg being firmly bandaged on to the splint in order to keep the foot at right angles. Care must, however, be taken not to produce sores by too tight bandaging, yet, on the other hand, the heel is very liable to slip upwards on the splint unless it is firmly held down by binding the foot-piece. Sometimes such bandaging to keep the foot at a right angle may be too painful to be borne, and it will be necessary to allow a certain amount of foot-drop, with the risk of secondary gastrocnemius contracture. Contracture of the hamstrings should be treated in the early stages with extension by weights attached over pulleys at the foot of the bed either to leather anklets or to the rectangular splints. If flexor contracture of the knees does not give way to this treatment, daily tension may be applied by means of a Macintyre splint, the inclined plane being gradually straightened out by a few turns of the screw daily.

Slight contracture of the gastrocnemii will disappear as soon as the patient begins to get about again on his feet, but occasionally tenotomy may be necessary to bring the foot to a right angle, or even for contracture of the hamstrings.

Prognosis.—Unless death occurs in the earlier stages from heart failure or pneumonia, or later from pulmonary tuberculosis or renal disease, complete recovery from

paralysis may result, unless alcoholism is resumed. In a severe case two years may elapse before full strength returns, though chronic mental weakness and loss of memory may persist.

Lead Neuritis

Chronic intoxication by lead is one of the more serious industrial diseases, occurring especially amongst painters, pottery glazers and those engaged in the manufacture of white lead. The latter are usually women, who unstack the white lead, the initial signs of plumbism being commonly anæmia and premature miscarriage. A blue line is usually present on the gums, which on examination under a lens seems to consist of bluish-black spots near the edge of the gum, with the exception of gaps due to missing teeth. If the gums are firm and the teeth kept scrupulously clean the blue line may be absent, as its presence is due to the formation of sulphide of lead in the gum by the absorption of sulphuretted hydrogen from decomposing food in contact with the teeth. In the potteries it is especially those whose work lies in applying a glaze to the ware who are in danger of lead poisoning, and for this reason various leadless glazes have been invented. Painters are frequent sufferers, and form the large majority of those affected by lead poisoning in London and most cities. Lead is absorbed into the system either by inspiring the dust of white lead or by swallowing minute portions through incomplete cleansing of the hands. Lead may, however, be absorbed in many other ways. The water supply may be a source of contamination, and for this reason leaden cisterns and considerable lengths of lead piping should never be used, especially if a peaty water is provided, the acids in it dissolving the lead very easily.

One of my patients when formerly living in the north of England had suffered for weeks from epigastric pain, diagnosed by his medical attendant as duodenal ulcer. Operation was arranged for, and this would have been

performed had not his butler on the previous day developed double wrist-drop, which was recognised to be due to lead neuritis. This led to a revision of the diagnosis of duodenal ulcer and an investigation of the water supply, which brought to light the fact that the main water supply which had recently been laid on to the house had been brought up the avenue in a leaden pipe instead of by iron pipes, as contracted for.

Beer, too, may become contaminated from standing overnight in the leaden pipes that carry it from the cask in the cellar to the engine at the bar.

Cider-drinkers, too, have been known to suffer excessively from the liquor brewed in leaden vats, both in Somerset and also in Poitou, hence the term *colica Pictonum*. Children may absorb lead and develop acute lead poisoning by smearing their hands with paint through swinging on painted wooden rails, a common cause in Australia. Chronic lead poisoning is also a cause of arteriosclerosis, gout and chronic renal disease. Albuminuric retinitis is frequent, and even intense optic neuritis is met with. Lead colic is usually an early symptom, causing intense grinding pain in the abdomen, associated with obstinate constipation; these symptoms are apparently due to muscular tetanus of the colon, and may be relieved by a full dose of opium in association with a brisk purge, *e.g.*, half a drachm of laudanum with an ounce of castor oil.

Acute lead poisoning occasionally results from women drinking an infusion of diachylon plasters in order to procure abortion. I have seen very severe lead neuritis from this cause.

Lead neuritis or *saturnine palsy* is a degenerative neuritis practically limited to the motor nerve fibres, in the large majority of instances only those muscles supplied by the posterior interosseous nerve being affected. Weakness of the grip is noticed and soon inability to extend the wrist and fingers follows. This is mostly symmetrical, but in certain cases one forearm only is affected, usually the right,

this being probably due to the extra muscular strain on the working arm in painters.

In the slighter cases of wrist-drop it will be found that the extensors of the fingers may be very weak, causing inability to extend the hand with the fingers straight out, this action being entirely produced by the extensors of the fingers. In such partial cases the extensors of the wrist may escape, so that with the fingers flexed the wrist can be fully extended; in these cases the power of grip may be comparatively slightly impaired. In more severe cases, however, the extensors of the wrist also suffer, giving rise to complete wrist-drop, though usually the extensor of the metacarpal bone of the thumb escapes, as does also the supinator longus. Occasionally the extensors of the fingers are paralysed unequally, often the two middle fingers being more dropped than the first and fourth, while in other cases the index finger is less involved than the remainder. A swelling known as Gubler's tumour, probably synovial, is often noticeable at the back of the carpus. There is no atrophy of the intrinsic hand muscles, except in cases where an additional subacute poliomyelitis produces atrophy of the thenar and other hand muscles; this additional atrophy may or may not be symmetrical, and is not recovered from, although the wrist-drop, being a neuritis, ultimately gets well.

A so-called upper arm type of lead paralysis is sometimes met with in which deltoid paralysis is pronounced. There will be wasting of these muscles, and also of the supra- and infra-spinatus, and to a less extent of the biceps. This form is said to be common in men who grind the lead carbonate in a mortar.

Sensory symptoms are conspicuous by their absence, and there are no trophic affections of the skin, finger joints or nails, as are common in other forms of neuritis in which the sensory nerve fibres are involved.

Cramping pains in the calves sometimes occur, though the legs otherwise escape entirely almost always, except in children in whom foot-drop occurs as well as wrist-drop.

The muscular wasting of the extensors of the forearms is characteristic, the supinator longus showing up prominently against the marked flattening on the back of the forearm.

The electrical reactions will be those to be expected in an acute or subacute motor neuritis, full reaction of degeneration being present in the more paralysed cases, with partial R.D. in the slighter forms. In those cases in which the weakness appears to affect one forearm only, usually the right, it may be possible to demonstrate electrical changes in the corresponding muscles on the other and apparently unaffected extensors, such as a diminished faradic excitability and slight hyperexcitability to galvanism.

Paralysis of the trunk muscles or of cranial nerves is rarely seen in lead neuritis, though I have met with a case of bilateral abductor paralysis of the larynx; this condition is said to be especially common in horses working in lead mines.

A generalised progressive muscular atrophy is a rare sequel to lead poisoning, but it is not due to neuritis but to degeneration of the anterior horn cells in the spinal cord.

Prognosis and Treatment. — Severe cases of wrist-drop will take many months for recovery, sometimes even as long as two years, though ultimate recovery is almost assured. Absolute cessation of contact with lead in all forms is essential, so that the painter must give up his work. This is the chief difficulty in treatment, as it is usually impossible to persuade the worker to change his employment and to learn a different trade. Once a sufferer from lead neuritis, the nerves are hypersensitive to the poison, and recurrence of paralysis is almost certain if the former work of painting or pottery glazing is resumed. Daily massage and treatment of the paralysed muscles by labile galvanism will help to keep the muscles from excessive wasting, and will thus hasten recovery when the new nerve fibrils have grown down into them. A point of some importance is to keep the wrists in a position of semi-extension, in order to prevent constant over-stretching of the paralysed

muscles. This may be done by means of an elastic glove extension, which is fully described on p. 92.

This is far better than fixing the forearm and wrist with a so-called relaxation or cock-up splint, to which the fingers and wrist are bound in the extended position, as such an apparatus prevents all use of the hand, and when fixed for too long and too tightly in extension may give rise to severe arthritis of the wrist, leading to adhesions and permanent crippling. Indeed, during the war I saw many such bad results from over-zealous use of so-called relaxation splints in cases of wrist-drop from musculo-spiral paralysis.

Electrical treatment is undoubtedly of value in cases of severe paralysis from neuritis. When reaction of degeneration is complete, that is to say, loss of all faradic excitability, with sluggish contractions to reversals of a galvanic current, especially at the anode, it is, in my opinion, of very little use to employ the faradic current in treatment, although I am aware that some authors recommend it. Slow reversals of galvanism, as by a metronome reverser, or regular interruptions of the current by stroking the limb with a disc electrode, will after a few days' treatment of twenty minutes at a time often remarkably augment the strength of contractions obtainable to a current which at the commencement gave perhaps only poor results.

In addition to local treatment of the paralysed muscles, continuous administration of strychnine is valuable in all cases of muscular paralysis. For neuritis this may be given hypodermically, one-fifteenth of a grain twice daily, or one-twenty-fifth of a grain in a mixture three times daily after food. The internal administration of iodide of potassium in chronic lead poisoning is of doubtful benefit for promoting excretion of lead from the tissues, as analyses have not demonstrated any corresponding increase in excretion of the metal. On the other hand, cases have been recorded, and I have myself seen an excellent example of this, in which slight lead neuritis with weakness only of

the extensors of the fingers became acutely serious, with total wrist-drop, within two or three days of commencing the iodide of potassium. Such cases have been explained by the theory that the iodide combines with the lead which is present in the tissues as albuminate of lead, thereby flooding the blood vessels, and therefore the nerves, with an increased quantity of lead iodide. If this were the case, however, an increased excretion of lead should occur in the urine, but analyses do not support this.

Diphtheritic Neuritis

Occurring usually from three to six weeks after the sore throat, the first symptoms of post-diphtheritic neuritis appear usually in the region of cranial nerves, weakness of the soft palate causing nasal speech and nasal regurgitation of liquids, and loss of accommodation from paralysis of the ciliary muscles. Squint, too, due to external or internal rectus palsy is common, while the pupil reaction is often sluggish, though by no means fixed to light. The pharyngeal constrictors may also be weak, increasing the difficulty in swallowing, and abductor paralysis of the vocal cords in addition to the palatal palsy will indicate vagus involvement. Facial paralysis on one or both sides is not rare, and even the muscles of the tongue may show wasting and fibrillary tremor. Later, limb paralyses may develop of the type already described under alcoholic neuritis, the extensors of the feet and ankles suffering most, causing foot-drop and a " steppage " gait.

Paralysis of the respiratory muscles is common in severe diphtheritic neuritis, affecting either the intercostals or the diaphragm, though both may suffer in sequence. The heart's action may indicate a further sign of vagus involvement, becoming irregular, sometimes much slowed in rhythm—bradycardia—or the heart beat may be accelerated to 120 per minute.

Either of these conditions is a dangerous sign, indicating need for careful treatment. The patient should be kept flat in bed, and not allowed to sit up for any purpose, while injections of strychnine and atropine are given. In

other cases, the heart escapes, and there may be no muscular weakness of the legs, though pains in the legs are followed by considerable anæsthesia of the feet and legs up to the knees, with an ataxic gait. Though at first the knee jerks may be exaggerated they soon become lost, and the clinical picture then is one of diphtheritic pseudo-tabes. Such symptoms will improve gradually with massage and faradism to the limbs, though the knee jerks may remain absent for many months or even a year.

Rarely isolated nerves may be paralysed after diphtheria, without any general polyneuritis. Thus, I have seen paralysis of the serratus magnus muscle on one side, due to neuritis of the long thoracic nerve, follow a period of tachycardia after diphtheria.

Occasionally diphtheria may be contracted upon an open wound or upon a mucous membrane, or in new-born infants occasionally the navel may become infected with this disease. In such cases it has been observed that subsequent neuritis differs in type from that following an ordinary faucial diphtheria, in which cranial nerve complications are common, whereas in wound diphtheria the subsequent neuritis shows no predilection for cranial nerves except for paralysis of accommodation. Muscular paralysis is liable to affect first the muscles in the neighbourhood of the infected wound, the toxin probably reaching the nerve fibres through the perineural lymphatics, a process resembling an ascending neuritis. Orr and Rows have demonstrated such an ascending path for toxins and organisms via the perineural lymphatics to the central nervous system.

Walshe drew attention to this point in numerous cases of wound diphtheria seen during the late war in Egypt and Syria.

This may be taken as evidence that the special liability to cranial nerve paralyses in ordinary diphtheria is due to the contiguity of the infected fauces to the cranial nerves, and not to any special affinity of the diphtheritic neurotoxin for cranial nerves.

Arsenical Neuritis

Polyneuritis may follow either acute or chronic arsenical poisoning. In the former white arsenic is the usual source of the poisoning, and may be taken either with suicidal intent, or it may have been administered in an attempt to murder. White arsenic is almost tasteless, and has been mixed with flour for poisoning cakes or pastry, and has been inserted into chocolate creams. Usually when large doses of four or five grains or more are administered by the mouth gastro-intestinal symptoms predominate, intense gastric pain, vomiting and diarrhœa being set up, thus causing rejection of the larger portion of the drug.

Two grains is the minimum dose that has been known to cause death, but much more has been taken and yet recovery followed. Another source of acute arsenical poisoning is intravenous injections of salvarsan or other arseno-benzol compounds, and acute nephritis or acute polyneuritis may follow some days after the injection, without any symptoms of gastro-intestinal irritation.

When a large dose of white arsenic is administered by the mouth, if the patient survives the acute irritative stage, symptoms of neuritis with weakness of the legs and arms may appear within a few days, or may be delayed for some weeks. A single large dose of the poison, either of arsenious acid or of the organic preparations, cacodylates or arseno-benzol, may thus produce polyneuritis.

More commonly chronic arsenical poisoning is the cause of the neuritis, the arsenic perhaps being given medicinally as in Fowler's solution for the treatment of chorea, lymph-adenoma, leukæmia and other conditions, or it may be given as sodium cacodylate by hypodermic injection. Thus I have known most severe polyneuritis with bilateral wrist and foot-drop follow the administration of fifteen drops of Fowler's solution three times a day for six weeks, and severe paralysis and wasting of the legs, with intense burning pains and anæsthesia, followed injections of

sodium cacodylate, a total of forty grains being given in eighteen days in the treatment of chronic septicæmia. Chronic arsenical poisoning by repeated administration of small poisonous doses of white arsenic in the shape of weed-killer, with murderous intent, is also well known now as a cause of neuritis. At the end of the last century the well-known epidemic of polyneuritis in Manchester already referred to (p. 13) was traced to arsenical contamination of the beer.

The type of polyneuritis of arsenical origin closely resembles that due to alcohol, the symptoms usually commencing with pains in the limbs and numbness of the feet and hands, with subsequent wrist-drop and foot-drop, and rapid muscular wasting of the extensors in the legs and forearms. Cutaneous anæsthesia of the periphery of the limbs is usually more marked than in the alcoholic type, and the pains may be more severe and burning in character, and hyperæsthesia is sometimes intense, but the mental symptoms that are common in alcoholic neuritis are much rarer. Sometimes ataxic gait is a more pronounced symptom than foot-drop due to muscular weakness, and being associated with pains in the legs, anæsthesia and loss of knee jerks, a clinical picture resembling tabes is produced—"arsenical pseudo-tabes." The knee jerks are not invariably lost even when wasting of the legs below the knees and anæsthesia of the feet are very pronounced, and the same persistence of the knee jerk is sometimes seen in rheumatic polyneuritis.

In chronic arsenical poisoning gastro-intestinal disorders, pain, vomiting and diarrhœa will probably be the first signs to draw attention to the presence of a gastric irritant poison. Pigmentation of the skin is common in these cases, brown freckles appearing on the chest, abdomen and back, and indeed the whole body except the exposed face and hands may become pigmented almost to the colour of chocolate. The distribution of the pigment is very different from that met with in Addison's disease, in which it is especially the exposed parts, face

and hands, which show the deepest pigmentation. Thickening and exfoliation of the palms of the hands, so-called keratosis, is also met with, and herpes zoster is a well-known complication in the chronic administration of arsenic (see p. 290).

The prognosis in arsenical neuritis is good as soon as the source of the poisoning has been eliminated, though when extensive muscular paralysis, with wasting and degeneration, has ensued, many months, perhaps two years, must elapse before complete recovery occurs.

The treatment is the same as that already described under alcoholic neuritis, contractures being guarded against, and massage, electrical treatment and injections of strychnine being persisted in for many weeks.

Diabetic Neuritis

Since 1884, when both Marinian [32] and Bouchard [6] pointed out the loss of the knee jerks in diabetes, evidence has accumulated of the clinical types of the nervous complications and of the pathological changes in this disease.

Davies Pryce [11] and Pavy in 1887, and later Williamson [51] in this country wrote on diabetic neuritis, while in 1888 Leyden classified the clinical forms of this disease into three types: (1) Hyperæsthetic, or neuralgic, (2) Motor, or paralytic, (3) Ataxic, or Pseudo-tabes. This is, perhaps, too artificial a division, which may be simplified into two forms, sensory and motor types, though these, too, constantly overlap.

Neuritis in diabetes is uncommon under the age of forty, though it must be remembered that in many subjects of glycosuria alcoholism may be the predominant factor, and the neuritis may really be â potu and not diabetic in origin. It is more apt to occur in the degenerative period of life, when at the same time vaso-motor and arterio-sclerotic changes are progressing, often leading to the terminal stage of gangrene in the toes and feet. Severe

pains in the feet may precede for weeks the œdema, erythemata and cyanosis that lead on to gangrene, and though vaso-motor neuritis may be concerned in the development of this condition, endarteritis obliterans is the primary factor, and the absence of arterial pulsation in the dorsalis pedis and posterior tibial arteries will aid in distinguishing this most serious condition from diabetic neuritis.

In the latter disease dull aching pains in the legs may persist for weeks or months, the pain being often severe enough to prevent sleep.

Cramps in the calves are a frequent sign, and the muscles of the legs may be tender on pressure. Both knee jerks and Achilles jerks may be present at this stage. Later, burning sensations may be complained of, and hyperæsthesia of the skin of the toes, feet and legs, with tenderness to the lightest touch, so that one leg cannot be borne upon the other, or even the weight of the bed-clothes on the legs may be insupportable.

Some of the deep reflexes, especially the Achilles jerks, may now be lost, and numbness and tingling of the feet and legs develop.

At this stage the sufferer may complain of tenderness of the soles of his feet, and of a sensation as if he were walking on pebbles, and perforating ulcer of the foot may be found. In a few cases these symptoms may progress to the stage of muscular paralysis, with loss of knee jerks, bi-lateral foot-drop and anæsthesia of the legs and wasting, as in alcoholic neuritis. This is, however, very rare in diabetes. More common is the limitation of the neuritis to the sensory nerves, which may progress to the stage of ataxy, the gait then resembling that of locomotor ataxy, or diabetic pseudo-tabes, not the steppage gait of foot-drop.

Frequently isolated nerves are attacked by the neuritis, such as the external popliteal, anterior crural, musculo-spiral or ulnar, or one of the motor nerves supplying the eye muscles. Pain is usually severe in these cases,

lasting for many weeks, though as a rule no muscular paralysis occurs and the pain is liable to be mistaken for sciatica or rheumatism. Uni-lateral loss of Achilles jerk may result if the posterior tibial nerve is attacked, while anterior crural neuritis, a characteristic manifestation of the diabetic toxin, is followed by loss of the knee jerk on that side. As a rule, no detectable anæsthesia results from such neuritis, though I have seen hyperæsthesia of the front of the thigh and inner side of the leg in anterior crural neuritis. In another case I saw severe sciatica of the right limb in a chronic diabetic, *æt.* 60, causing intense pain, develop to the stage of complete foot-drop, though the paralysis ultimately recovered.

Ocular palsies, producing diplopia, are sometimes met with from neuritis of either the third, fourth or sixth cranial nerves. These symptoms will be preceded by severe hemicrania, or uni-lateral neuralgic pains referred behind the eye and forehead to the temple and back of the head.

These neuralgias may be periodic for many hours in the morning, clearing off in the afternoon. Slight proptosis may accompany the ocular palsy, indicating a congested and œdematous condition at the back of the orbit, and may persist after the ocular paralysis has cleared up. Of 58 cases of ocular palsy collected by Dieulafoy,[12] 35 were of the sixth pair, or simple external rectus palsy, but total third nerve paralysis may be met with. The onset of the paralysis is usually somewhat sudden, clearing up after a few weeks, though recurrence is said to be not uncommon.

Reflex iridoplegia, or Argyll Robertson pupil, I have seen in diabetic neuritis, as well as in chronic alcoholism, with no evidence of syphilitic infection, and a uniocular Argyll Robertson pupil may be the only souvenir of a previous attack of third nerve paralysis. Sometimes the pupil in diabetics is small and may be seen to react to strong light, though sluggishly, as in morphinism. Loss, or much diminution of the pupil reflex to light, when

present in addition to other signs of diabetic pseudo-tabes, such as pains, ataxy, numbness and anæsthesia, loss of the deep reflexes, and perhaps perforating ulcer and a history of ocular palsy, makes the resemblance to true tabes so close that considerable difficulty may be found in distinguishing the two diseases. Blood Wassermann tests and examination of the cerebro-spinal fluid may give doubtful results, and the presence of glycosuria may indeed alone decide the diagnosis.

Diabetic retinitis is a well-known complication of the disease, but retro-bulbar optic neuritis I have never met with, nor have I seen any reference to it, though it is a condition that might well be expected to occur.

Morbid Anatomy and Pathology.—Isolated diabetic neuritis is an inflammatory perineuritis, leading to interstitial neuritis, and in a few cases to considerable degeneration of the nerve fibres. In the symmetrical cases, parenchymatous degeneration of the medullary sheaths probably occurs first, before swelling of the fibrous sheaths takes place. In some cases degenerative changes have also been found in the posterior roots, with secondary ascending degeneration in the posterior columns. Nerve-cell degenerations in the anterior horns have also been described. Such are the morbid changes that have been found, but their explanation and pathogeny is a difficult problem. They can scarcely be due to a mere excess above normal of the blood sugar, neither can acetone nor diacetic acid be fairly blamed, as neuritis may occur in the absence of both these substances. An attractive theory at first sight is their attribution to an endocrine deficiency, owing to sclerosis of the islands of Langerhans in the pancreas. This lack of internal secretion, of the nature of insulin, might thus be considered to be the cause of diabetic neuritis, a process somewhat comparable to the production of the tropical disease beri-beri, a form of polyneuritis due to vitamin deficiency. On reflection, however, this theory of endocrine deficiency as the cause of diabetic neuritis appears to be untenable, for it does not explain

the transient nature of the neuritis, recovery after a few weeks or months being the rule. This could not take place if endocrine deficiency were the cause, nor is it easy to understand how any deficiency cause could produce an asymmetrical inflammation and degeneration of an isolated nerve. The temporary and recurrent nature of the attacks is certainly more suggestive of a varying degree of some toxic factor in the diabetic blood.

A deficiency cause, such as the endocrine deficiency in diabetes and the vitamin deficiency in beri-beri, can act indirectly only, by setting up changes in the metabolic processes of the tissues, thus probably giving rise to some insufficiently broken-down excretory product, which in the course of circulation affects the peripheral nerves, causing parenchymatous degeneration. Why a circulating toxin should sometimes set up an asymmetrical neuritis is not always easy to understand; it is usual to explain it by inferring a locally acting cause in addition, such as overuse or strain of the limb, as when the right forearm is affected alone in painters, or by undue pressure on the nerve. In the asymmetrical neuritis of rheumatism, septicæmia, or paratyphoid fever, the asymmetry may be due to local involvement of the nerve sheaths by direct organismal infection, as in leprous neuritis, but in diabetes no organisms are known to be at work.

Treatment of Diabetic Neuritis.—When hyperæsthesia of the limbs is present, massage, electricity and strychnine must be avoided. As the general tendency for this form of neuritis is towards recovery after several weeks, or even months, little anxiety need be felt on that score, while aspirin, phenacetin, caffeine and small doses of medinal will be useful in controlling the neuralgic pains and in inducing sleep.

The most important part of the treatment will be general dietetics and the question as to the advisability of using insulin. So long as glycosuria persists the use of insulin by hypodermic injection in doses of ten to twenty units twice daily will not be dangerous, while it may be

beneficial in causing the disappearance of headaches, or hemicranial or other neuralgic symptoms, with corresponding improvement in the general health.

Beri-beri

Beri-beri was formerly a very common disease in India, the Straits Settlements and Japan. At one time it was thought to be an infective disease, by some ascribed to a fish diet, by others to bacterial infection, the result of defective sanitation. Formerly it was rife in the Japanese Navy, and its eradication was ascribed to modern methods of hygiene introduced. Of late years the increasing knowledge of vitamins, and especially experimental work in the production of avian polyneuritis by feeding pigeons on polished rice, and later curing the disease by the administration of watery extracts of the husk, have shown beri-beri to be a deficiency disease, the degenerative polyneuritis being produced by the deficiency of vitamins in the polished kernel of the grain. Practically all the vitamins are contained in the husk of the rice grain, so that the highly polished rice produced by the modern mills is no longer a complete food, but must be supplemented by other sources of vitamins such as fresh fruits, milk, meat and vegetables, etc.

Two clinical types of the disease are recognised, the so-called wet and dry forms. In the former œdema of the trunk and limbs, due to cardiac failure, may be a pronounced symptom, the heart being dilated and rapid in its action. In the so-called dry form œdema may be absent, and the heart not obviously affected, but muscular wasting may be extreme in the legs and arms, with complete foot-drop and subsequent contractures.

Malarial Neuritis. Rarely seen in this country, polyneuritis resulting from malaria has been many times reported, and it may be symmetrical in the extremities or may cause local asymmetrical atrophy.

Dysentery of bacillary origin may be followed by severe

and chronic bilateral sciatica, though a generalised multiple neuritis is rarely seen after this disease.

Enteric and Paratyphoid Neuritis. Here again asymmetrical neuritis is common, severe pains around the shoulder preceding atrophy of the trapezius or of the serratus magnus, or of the rhomboids and levator anguli scapulæ, in neuritis affecting the spinal accessory, long thoracic and posterior scapular nerves respectively after paratyphoid.

I saw a man of 29 with paratyphoid, who, a week after admission to hospital, developed severe pains in the upper arms and shoulders, which kept him awake, and were followed by marked paresis of the right serratus, latissimus, triceps and extensors of the right fingers and thumb, and of the left deltoid.

Another case of asymmetrical neuritis of the right median, ulnar and musculo-spiral nerves I saw in a man aged 27, as the sequel of a febrile attack of uncertain origin with tonsillitis, followed by acute nephritis.

There was never any peeling of the skin to suggest scarlet fever, and bacteriological tests for diphtheria, enteric and paratyphoid were negative. Practically complete paralysis of the arm and hand resulted, with atrophy of the finger pads and skin, though complete recovery took place in two years. Possibly the infection was rheumatic, as nephritis is one of the rarer complications of this disease.

In such cases none of the generalised symptoms of polyneuritis are present, the knee jerks being unaffected, and no numbness of the lower limbs, foot-drop or ataxic gait are evident. Polyneuritis has also been described after many other infections such as measles, whooping cough, scarlet fever and parotitis. Heerfordt, Feiling[15] and others have described a form of polyneuritis as a complication of a form of parotitis which is associated with uveitis. Facial palsy, and sometimes other cranial nerve paralyses, and neuritis of the limbs may occur.

Septicæmic Polyneuritis

Streptococcal infection with suppuration following slight abrasions of the surface is not very rare, and uterine infection following miscarriage or pregnancy may similarly cause polyneuritis. This should not be called puerperal neuritis, with which it has often been confounded. Septicæmic polyneuritis resembles the alcoholic type and I have seen it associated with intense pain, tenderness and hyperæsthesia of the skin. More usually the sensory phenomena are limited to tingling and numbness of the extremities with pins and needles. Wasting of the finger pads, glossy skin and trophic wasting of the nails may also result. In one case where slight suppuration in the ball of the thumb followed a graze of the skin due to a fall from a bicycle, the only symptom three weeks after the injury was loss of power of accommodation, suggestive of diphtheritic neuritis, though there was no other reason to suggest wound diphtheria, the skin wound having healed up in a week.

Similar loss of accommodation was present for a month in another case of septicæmic neuritis following infection of the foot, with formation of an abscess.

A youth, *æt.* 19, early in June 1896 picked a soft corn off the ball of his right foot. Two days later the foot began to swell and became painful, the swelling soon spreading up to the knee. Five days later an abscess in the ball of the foot was opened, and enlarged glands were noticed in the groin. The swelling did not subside for four weeks, and then he noticed numbness of the other knee, which gradually spread down to the left foot, then attacking the right foot and spreading up above the right knee. Five weeks after numbness appeared in the feet he noticed numbness in the hands, which soon became weak so that he could not do up buttons, and the wrists were slightly dropped. His legs became steadily weaker so that he was unable to stand, no improvement occurring until four months after the commencement. For a month he was quite unable to read. The knee jerks were lost, the feet cold and sweaty and dorsiflexion of the ankles

very weak. There was never any diplopia or nystagmus. He complained of no pain except tenderness of the calves, and there was no anæsthesia to be detected, except slight numbness of the soles. Recovery was complete.

In another case the neuritis followed uterine infection, and was ushered in by nasal regurgitation and difficulty in swallowing.

A girl, *æt.* 24, had four or five times produced artificial abortion in herself by inserting a sharp instrument. In September 1896, after another abortion, she developed septicæmia and peritonitis, with constant vomiting, pains in the stomach and constipation. Recovering from this, a month later she had difficulty in swallowing, with choking and nasal regurgitation, which lasted for over three months. Her legs became weak and she was unsteady on her feet soon after the difficulty in swallowing appeared, with pains in her heels, spreading up into the calves, with dropped feet. Then tingling appeared in the fingers and the hands became weak, the left wrist dropping. Two months after the onset the pains in the legs became very severe, and when I first saw her in February 1897 the tenderness of her legs was so extreme that they had to be swathed in thick cotton wool. The feet were much dropped, and the legs and forearms were much wasted, the finger tips being atrophied and the skin glossy and wet with perspiration, while her power of grip was nil, the left wrist being dropped. The knee jerks were absent, and lateral nystagmus in both directions was noticed. Alcoholism was certainly excluded as the cause. She improved rapidly under treatment; as soon as the hyperæsthesia and tenderness diminished she was treated by massage, galvanism and strychnine injections. She insisted on returning home after a month's treatment, against advice, and died suddenly two days later, probably from heart failure.

Septicæmic neuritis may occasionally be asymmetrical, and may affect a nerve trunk close to the intervertebral foramina, as in the case of a gunshot wound of the left arm, followed four weeks later by pains in the opposite arm and right side of the neck, with paresis of the right

serratus magnus and winged scapula, with hyperæsthesia of a cutaneous strip on the ball of the thumb and along the outer forearm extending up to the shoulder, suggestive of neuritis of the sixth cervical nerve.

Chronic septicæmia, due to dental or intestinal sepsis, is a frequent cause of fibrositis and neuro-fibrositis, in which vague irregular pains occur around the shoulders, back and limbs. In some cases, the local fibrositis, in addition to involving sensory nerve endings, may spread to larger nerve trunks and may involve the brachial plexus or the sciatic nerve, setting up brachial neuritis or sciatica. Rarely a true multiple symmetrical neuritis may also be caused by this form of chronic septicæmia.

Serum Disease

The use of anti-streptococcal and anti-typhoid sera has been stated to cause polyneuritis, and cases have been published in which repeated injections of an anti-serum have been followed by symmetrical pains and tingling in the extremities and even muscular paresis. The difficulty, however, at once arises of deciding whether the septicæmia or typhoid on the one hand or the anti-serum should be held responsible for the polyneuritis. During the War, when such vast numbers of men were treated with anti-tetanic serum, in the course of my work as one of the two tetanus inspectors for the London area, and as a member of the Tetanus Committee, I came a great deal into contact with tetanus cases and the routine use of anti-tetanic serum, both for wounds and in the treatment of tetanus, yet I saw no case of polyneuritis follow in any case of anaphylaxis or other phenomena due to serum treatment. Moreover, should polyneuritis follow recovery from tetanus, the difficulty would arise of allowing for the effect of the septicæmic infection which invariably accompanies infection by tetanus.

Certainly post-diphtheritic neuritis should be held to be due to the diphtheritic neurotoxin, and not to any anti-diphtheritic serum employed in the treatment. Probably

the same argument should apply in the cases of septicæmia, tetanus and typhoid when treated by serum injections.

Puerperal Neuritis

Polyneuritis complicating the puerperium may occur either during the course of gestation, or more commonly within a few weeks of childbirth.

Some authors ascribe these cases invariably to septicæmic infection, explaining those which occur before child-birth as being due to absorption of toxic products remaining in the uterus from a previous pregnancy. This explanation I cannot accept, and unless other signs of septicæmic infection are evident after delivery, such as pyrexia, septic discharges, etc., another source of the polyneuritis must be looked for.

Possibly it is due to a temporary endocrine deficiency associated with the profound metabolic changes consistent with gestation. Puerperal neuritis is usually not a painful form, numbness and paræsthesiæ predominating, with weakness and loss of knee jerks. In one case I saw the patient had, in addition to generalised polyneuritis, facial diplegia and diaphragmatic paralysis, though complete recovery ensued in three months.

A case of recurrent polyneuritis affecting the flexors of the hips during pregnancy was published in 1895 by Ormerod.[35]

A woman, *æt.* 31, on four occasions and always at the fifth month of pregnancy developed weakness of the iliopsoas on each side, so that she was unable to walk upstairs, to cross her legs, or to rise from the kneeling position. At the same time as the weakness of flexion of the hips appeared she used to suffer from pains in the back and legs. The weakness and pains persisted during each pregnancy until two months after the child was born, when she recovered, with the exception of the last occasion. Eighteen months afterwards the paralysis of the flexors of the hips was still complete, and she was unable to rise from a kneeling position or to cross the legs. The knee jerks were absent, but the extensors of the knees were

powerful, and no other muscles appeared to be affected, except the shoulders for a time. She recovered completely after four years.

It should not be forgotten that the act of delivery, especially if aided by forceps, may cause pressure upon nerve trunks within the pelvis, usually the obturator, anterior crural or great sciatic nerve of one side being damaged.

In one case I saw sciatic paralysis with numbness of the foot and foot-drop on one side, complicated by musculo-spiral paralysis with wrist-drop in the arm on the opposite side, follow delivery by instruments under an anæsthetic. This was an excellent instance of pressure paralysis produced in two different ways, the sciatic being damaged within the pelvis during delivery, while the musculo-spiral palsy was due to faulty position of the arm against the sharp edge of the table during the anæsthetic. The diagnosis with which this case was sent up to the hospital was "crossed paralysis," whatever that may have been supposed to mean.

Carbon Monoxide Neuritis

It is said that a single severe dose of poisoning by carbon monoxide may be followed by neuritis, as may also occur in arsenical or lead poisoning, an unusual feature in polyneuritis, which is usually the sequel of a more sustained or chronic toxic absorption. Care must, however, be taken in cases of carbon monoxide poisoning not to mistake pressure neuritis of the musculo-spiral, external popliteal or other nerve trunks for toxæmic neuritis, pressure palsies being especially liable to occur in persons rendered unconscious by coal-gas poisoning, or in explosions of fire damp in coal pits through lying on a hard surface in one position for many hours.

Carbon monoxide is also produced by the incomplete combustion of petrol vapour, and may be a serious danger from running a petrol engine in a closed garage or other unventilated space.

Carbon Bisulphide

Polyneuritis due to inhalation of fumes of this drug may occur in badly ventilated boot workshops from the work of solutioning on rubber soles.

Headaches for some months are a premonitory feature, occurring towards the end of the day's work and disappearing by the following morning. Bilateral paresis of the dorsiflexors of the feet and ankles and of the extensors of the fingers and wrists will be accompanied by wasting and electrical reaction of degeneration. Wasting of the intrinsic musculature of one or both hands may occur. In one case that I have seen, although the muscular paresis was marked in the feet and hands, the knee jerks were normal throughout, and there was no trace of any sensory involvement, there being no pain or numbness at any time.

Tuberculous Neuritis

Alcoholic neuritis is frequently complicated by pulmonary tuberculosis, but the neuritis is doubtless mainly alcoholic and not tubercular in origin, though the doubly acting causes of alcohol plus tubercle may combine to produce the polyneuritis, just as alcohol and arsenic together may produce polyneuritis, when neither alone is in sufficient quantity to produce this effect. Apart from such doubly acting causes, tubercle is a very rare cause of neuritis, and I have seen only one case of generalised polyneuritis apparently due to miliary tuberculosis, in which no other cause of neuritis was ascertainable.

Tuberculous Neuritis of the Feet.—In two cases of severe and fatal tuberculous infection I have seen symmetrical pains and wasting of the intrinsic musculature of the feet, with glossy skin. The first case had been diagnosed as gout, in spite of extensive pulmonary tuberculosis, by a well-known physician in 1895, and remembering this case when I saw the identical condition of the feet seven years later in a woman with obscure renal

disease and pyrexia, I confidently diagnosed tubercle as the cause, which was confirmed at the autopsy.

Syphilitic Neuritis

Syphilitic infection occasionally produces generalised polyneuritis, though this is a rare complication. According to Pétren, true syphilitic polyneuritis occurs only within the first year after infection.

Polyneuritis occurring later may be due to other causes, especially to excessive arsenical or mercurial treatment.

In some of the reported cases the neuritis appeared to be associated with anterior horn-cell damage, as atrophy of certain groups of muscles such as the anterior tibial remained permanent. Symmetrical neuritis of the seventh and eighth cranial nerves is a well-known, though rare, complication in the secondary stage of syphilis. The clinical picture is that of double facial paralysis with bilateral nerve deafness (see pp. 366, 374).

Malignant Disease.—Carcinoma undoubtedly occasionally produces polyneuritis. Possibly a degenerative neuritis due to cachexia, more probably the polyneuritis is due to septic absorption from abscess cavities in connection with the growths, which usually involve the stomach or colon in these cases. In one case I saw secondary to carcinoma of the colon, which had lasted seven years at least, and ultimately developed large secondary masses in the liver, the polyneuritis was limited to the upper extremities, intense pains in the shoulders, arms and forearms being complained of, "like a rat gnawing." The distribution of the muscular paralysis too was unusual, there being complete paralysis of both triceps muscles and of the extensors of the fingers and ulnar extensors of the wrists. The legs were quite normal and the knee jerks brisk.

Anæmia

Chronic anæmia is said to cause polyneuritis, though this form is much rarer than the spinal degeneration of combined sclerosis met with in conjunction with grave

anæmia of pernicious type. I have met with well-marked polyneuritis in a youth of 17 who died with symptoms of lymphatic leukæmia,[24] the peripheral nerves as well as the liver and kidneys being found to be infiltrated with lymphocytes. It is difficult to conceive that the symmetrical polyneuritis could be due to pressure of the lymphocytic exudation upon the nerve fibres: more probably it was of toxic origin in some way connected with the leukæmic process.

Senile Polyneuritis

In old people over the age of 70 polyneuritis, affecting especially the lower limbs, may occur in the absence of any recognisable toxæmia, and is by many suggested to be degenerative neuritis due to senility. If that were the case improvement could hardly be expected to occur, but under ordinary treatment by rest, massage, electrical baths, and strychnine, recovery may be practically complete. The neuritis is usually not very painful, mostly numbness and paræsthesiæ of the extremities, with loss of knee jerks and some weakness of the dorsiflexors of the feet.

It is important to go carefully into the question of the absorption of any known toxin productive of multiple neuritis; for example, I have seen polyneuritis develop in a man of 75 who was taking three small whiskies daily and no other form of alcohol, though this amount was an increase on what he had been previously accustomed to. Cutting off all alcoholic stimulant was followed later by a very great improvement, and it was clear that in this case a small amount of alcohol which would not have been sufficient to produce neuritis in middle life was capable at the age of 75 of setting up degenerative neuritis owing to lack of vitality in the nervous structures, thus justifying the diagnosis of senile polyneuritis.

Toxic Polyneuritis

As already stated, the term toxic polyneuritis is applied to forms of polyneuritis of unknown origin, or in which

the toxin is little understood. Under this group we may distinguish several distinct forms—the acute febrile, rheumatic, slow chronic, relapsing, and hæmatoporphyrinuric amongst others.

Possibly, if not probably, the toxin of acute febrile polyneuritis is bacterial, while an autotoxin of metabolic origin is probably responsible for other forms. An enterotoxin, associated with obstinate constipation or chronic indigestion, is apparently the basis of acute hæmatoporphyrinuria, but although we have vague indications of the pathogeny of a few types, yet the fact remains that large numbers of cases of acute polyneuritis are met with in which no indication of a cause can be found. Perhaps one of the most obscure types is the slow chronic polyneuritis, in which external toxins can be excluded.

The following may be taken as an example of toxic polyneuritis, in which no known cause of polyneuritis could be detected.

A man, *æt.* 30, a coachman, developed pains in the forearms, calves and thighs at night, with a general feeling of tiredness and a sensation of painful grip under each breast. He became short of breath, and his walking became difficult, owing to catching his toes. Ten days later left facial paralysis appeared.

The pains continued for several weeks, especially at night, waking him up, and being worse in the forearms. The knee jerks were absent, but there was no weakness of dorsiflexion of the feet or of extension of the wrists or fingers. The feet were sweaty, and there was numbness of the hands, especially of the pads of the fingers. No diplopia, but definite lateral nystagmus. No anæsthesia of the legs, but slight Rombergism was present.

There was no history of alcoholism, of sore throat, lead poisoning, chill or of any known cause of polyneuritis.

Amongst the acute forms of toxic polyneuritis must be classed the majority of cases of so-called Landry's paralysis. This elusive group of cases of ascending paralysis is not to be looked upon as a clinical syndrome with a morbid anatomy of its own, but with little doubt is made up of

cases of toxic polyneuritis, with a few cases of true ascending myelitis, and others undoubtedly acute poliomyelitis.

Acute Febrile Polyneuritis

Acute febrile polyneuritis was described in 1892 by the late Sir William Osler,[36] and again during the Great War Holmes,[26] Bradford [7] and others drew attention to this disease. The sequence of the polyneuritis to distinct malaise and pyrexia places this type definitely in the infective rather than in the autotoxæmic group, though the exact source of the toxin has not yet been worked out. Usually the disease is ushered in by a premonitory stage of a few days with malaise and chills, and from three to six weeks later signs of polyneuritis appear with pains in the back and limbs, hyperæsthesia, weakness and shakiness of the arms and legs. In many cases facial diplegia occurs, and there may be a feeling of intense depression, associated with exceeding weariness and a furred tongue. In those cases in which foot-drop occurs the knee jerks are likely to be lost, but the knee jerks may remain unaffected when the lower limbs escape.

Bacteriological examination of these cases has so far proved negative, but may be taken to exclude diphtheria and paratyphoid. Clinically, too, most of the infections that are known to produce polyneuritis, such as influenza, measles, scarlet fever, whooping cough, etc., may be excluded.

Rheumatic Polyneuritis

Acute rheumatic polyarthritis is unknown as a cause of polyneuritis, yet the latter is not an uncommon sequel of a sharp exposure to wet and cold. I have seen the most severe polyneuritis, with intense hyperæsthesia and tenderness of the limbs, follow shortly after a thorough wetting and chill in a woman of 50, who became drenched with rain through the thatched roof of her cottage giving way during a storm at night.

Another woman, *æt.* 36, suffered severely from chill for several days after exposure to cold wind and rain for many hours in December 1924. She continued to feel weak and ill until, seven weeks after the wetting, she noticed that her vision was becoming misty, and she could see nothing clearly, though she had no definite diplopia. A month afterwards her gait became very unsteady, and when I saw her a week later her gait was very ataxic, she was unable to raise her toes from the ground or to dorsiflex her ankles against resistance, the peronei muscles being completely paralysed, though the tibialis anticus had fair power. The knee jerks and Achilles jerks were absent, there was slight tenderness of the calves, and partial anæsthesia and analgesia of the feet and legs. She had never had any pain or tingling. Vision was much affected, she could not see my face; and central scotomata were found in each eye for 15° around the fixation point, using a white disc of $\frac{1}{2}$ inch diameter. There was no history of alcoholism, but syphilis had been acquired from the husband three years previously.

The vision improved after another week, the central scotomata completely disappearing, and the foot-drop also gradually recovered.

The chill three months before the development of the foot-drop, being succeeded by continued malaise, must be accounted as the main factor in the development of the polyneuritis, though the antecedent syphilis of three years previously may be held as a predisposing cause. How far actual "rheumatism" can be held accountable is problematic. The term rheumatism is very indefinite and elastic, though if it be held to include the effects of a "rheum" or chill, then this case may be considered to be a typical rheumatic polyneuritis.

Polyneuritis from chill may cause double facial paralysis without producing any muscular paresis of the limbs, and is to be distinguished from facial paralysis due to locally acting causes.

In April 1897 I saw a man, *æt.* 45, who ten weeks previously had been badly chilled by wearing a wet coat, followed two days after by severe aching pain in the lower part of his back, the pain keeping him awake at night.

After a week the pain shifted for another week to the centre of his back, then disappearing. Two days later, or 16 days after the chill, he woke up in the morning with the left side of his face completely paralysed, and after another two days the right side became paralysed also. Four days later pain gradually appeared along the insides of the feet, much worse in the left foot and at night, and for five weeks the pain was so severe that he could hardly stand. No other cranial nerve was affected in addition to the facial diplegia, and there was no muscular paresis of the limbs, nor any anæsthesia, and the deep reflexes were normal. Taste sensation was perfectly acute on both sides. Reaction of degeneration was present in the facial muscles of both sides.

The history of this case in some respects resembles that of many cases of acute febrile polyneuritis, the pains and facial diplegia being common to both, but there was no evidence of pyrexia in this case, and the history of exposure to severe chill was very definite.

Subacute rheumatic polyarthritis may be complicated by polyneuritis.

An unmarried woman, *æt.* 47, a school teacher, in 1921 had for some months suffered from swelling of the hands and of the left knee-joint, with pain on movement. She then began to suffer from burning pains in the legs, the feet becoming numb two months later, with loss of power of walking. She complained of pins and needles in the fingers, and developed finger-drop owing to paresis of the extensor communis, and weakness and wasting of the intrinsic musculature of the hands. There was tactile anæsthesia and analgesia of the left hand up to the wrist, but no objective loss of sensation on the right hand, though she said it did not feel natural. There was complete left foot-drop and some weakness of the right dorsiflexors. She could move the right but not the left toes. Though the right foot was much stronger than the left, there was much denser anæsthesia of the right limb, extending to just above the knee. The knee jerks were both brisk and equal, the Achilles jerks were absent, and the left forearm flexor jerk was also absent, though the right was brisk. After two months the pains in the legs increased considerably, and she complained of a curious

subjective sensation "as though she had several pairs of legs in bed."

This continued for several weeks. Recovery was slow but steady.

The term *Slow Chronic Polyneuritis* may be applied to cases in which the symptoms gradually increase during many months, and in which no known toxic agent is at work, such as alcoholism, lead or arsenic. Pain is usually either absent throughout, or only trifling at the commencement, and there may be extensive atrophy. The limbs may gradually become more and more paralysed over a period of twelve months, so that ultimately there may be total paralysis of all four extremities. The patient may be unable to sit up and there may be slight wasting and paralysis of the eyelids, lips and tongue. The deep reflexes are lost and in some cases there may be extensive atrophy of the dorsiflexors of the feet and hands, with reaction of degeneration. In a few cases sensation is considerably impaired, and in other cases there may be no sensory involvement, though then muscular atrophy is only slight, but the paralysis may be profound for a period of many months.

In the case of a man, *æt.* 31, the symptoms gradually increased during twelve months, commencing with pain in the calves and a tired feeling, after the appearance of some large purpuric blotches on the legs. The legs gradually became enfeebled to the stage of total flaccid palsy, the hands and forearms not being involved until nine months after the commencement of the symptoms. When I saw him seventeen months after the onset there was practically complete palsy of the feet and legs, hands and wrists, with contracture of the feet and hands. All the deep reflexes were lost, and there was considerable anæsthesia in the periphery of all four limbs. The sphincters were normal, and no cause suggestive of polyneuritis could be discovered. The Wassermann test was negative both in the blood and cerebro-spinal fluid.

A girl, *æt.* 15, became gradually weak in the legs and arms while at school in South Africa. After four months

she was sent to England for treatment, but continued to get progressively weaker during the next nine months, until all four limbs were totally paralysed: she could not raise her head from the pillow, the eyelids and lips were partially paralysed, there was weakness of the masticatory muscles and the tongue was somewhat wasted. No sensory symptoms were present at any time except slight paræsthesiæ of the fingers at the commencement. All the deep reflexes were lost, but only slight muscular wasting occurred, and no contractures. No improvement was observable in spite of daily massage and electrical treatment until eighteen months after the onset, when she slowly began to regain power, and ultimately recovered perfectly in another two years and has remained well for the past ten years.

The prognosis is usually good, especially in the type without sensory involvement, when complete recovery is likely to take place, though the whole course of the illness may last from two to three years. The slow development of the symptoms in such cases strongly suggests a continuously acting toxin rather than an acute poisoning, and one is driven to the supposition that an auto-toxæmia is at work. Certainly the continual absorption of external toxins may be excluded, as change of climate and even of country may produce no effect upon the slow progress of the disease.

Acuteness of onset of polyneuritis, however, is not sufficient reason to exclude an auto-toxæmic cause, as will be seen presently when dealing with acute hæmatoporphyrinuria.

Relapsing and Recurrent Polyneuritis

The commonest causes of recurrent polyneuritis are chronic alcoholism and lead poisoning, but recurrence is also met with rarely in cases where the cause is obscure. Thomson[49] records recurrent polyneuritis at the ages of 5 and 19 years in a youth whose second attack commenced five days after being thrown into the water, paralysis of all four limbs and both sides of the face becoming complete in another ten days. Sensory symptoms were very

slight, though considerable muscular wasting developed, and complete recovery occurred within two years. The acuteness of the onset of the paralysis following the immersion is strongly suggestive of chill being the exciting factor, though his previous similar attack at the age of 5 seems to demonstrate a predisposition in his metabolic processes to the development of a neuro-toxin, or possibly an excessive sensitiveness of his peripheral nerves to the action of such neuro-toxin.

In lead poisoning it seems to be established that a recurrence of wrist-drop and colic may be dependent upon some slight cause, as a febrile attack or an irregularity in diet, in the total absence of any fresh source of lead absorption.

This fact lends support to Mary Sherwood's suggestion that the first attack of neuritis leaves the nerves with a lowered power of resistance to the assault of a fresh toxæmia.

Grocco[19] was the first to describe recurrent attacks of polyneuritis in the absence of any obvious cause such as lead or alcohol. Other reports of such cases have been made by Eichhorst[14] and by Mary Sherwood,[43] who described two cases from Eichhorst's clinic in Zurich, though her first case was that of recurrent palsy of the right arm only, and should scarcely be classed with other cases of generalised multiple neuritis. Ross and Bury[40] described two sisters with recurring paralysis, and Targowla[47] recounts the case of a woman who had three attacks of paralysis at the ages of 19, 27, and 39 years. Sorgo[44] described the case of a man who after three attacks of abdominal pain and constipation, developed bilateral palsy of the arms, followed twelve months later by a recurrence of the paralysis and colic, and again eight months later, following a chill, he developed colic, severe pains in the limbs, paralysis of the arms, weakness of the legs, loss of the knee jerks, respiratory paralysis and death. Degenerative neuritis was found in the peripheral nerves. The sequence of the paralysis to attacks of severe abdominal pains and constipation is

suggestive of an enterotoxin having been the cause, and is strikingly similar to the mode of onset of the paralysis in acute hæmatoporphyrinuric polyneuritis.

H. M. Thomas [48] in 1898 published the case of a man *æt.* 28, who had five attacks of paralysis within six years. In his case chronic indigestion was a feature of the illness, and possibly here also an enterotoxin was the cause.

Nattrass [33] in 1921 described the case of a boy *æt.* 18, with double foot-drop, loss of the deep reflexes, and other signs of generalised multiple neuritis, who had three similar attacks at the ages of 4, 17 and 18 (see p. 54).

Recurrent polyneuritis has also been described in repeated pregnancies (see p. 35).

Polyneuritis with Parotitis

General symptoms of polyneuritis may accompany parotitis, with or without uveitis. Feiling [15] has drawn attention to the latter form, which is to be distinguished from mumps. After a period of malaise for some weeks, neuralgic pains and backache appear, followed by facial diplegia and irido-cyclitis and slight but firm swelling of the parotids. Patchy erythema may appear on the limbs, and slight generalised polyneuritis, with numbness and paræthesiæ of the extremities, and loss of the deep reflexes. Recovery is the rule.

Hæmatoporphyrinuric Polyneuritis

Polyneuritis associated with acute hæmatoporphyrinuria may occur as an acute illness in the absence of any poisoning by drugs such as sulphonal, trional, veronal, etc., or it may occur as a direct result of chronic poisoning by one of these drugs. To this latter group Günther [21] applies the term acute toxic hæmatoporphyrinuria. Sulphonal was first produced by the firm of Bayer in 1886, and the first case of hæmatoporphyrinuria resulting from its use was published by Stokvis [46] in 1889.

Since then numerous other instances have been put on

record. The first publication of polyneuritis with hæmatoporphyrinuria was by Ranking and Pardington [38] in 1890.

The acute symptoms are ushered in by severe pains in the abdomen and loins, constipation and sometimes vomiting. Dark brown to port wine-coloured urine is an early symptom, and the pains continue and may spread all over the trunk and limbs. The pulse-rate is often raised to 100 or 120, but usually there is no pyrexia. Retention of urine and even incontinence may occur, and occasionally mental symptoms may be noticeable, such as general nervousness, excitability, or even delirium and epileptic fits. Insomnia, too, is usually troublesome.

The symptoms of this variety of multiple neuritis vary, as is the case with other causes of neuritis. In some examples the sensory symptoms are more paræsthesiæ of the trunk and limbs than pain, while again in others the pain may be most severe and may be said to be, "like knives," or as if the flesh were being torn off the bones. Anæsthesia is present in some and absent in other cases. The stress of the multiple neuritis may fall either upon the upper or upon the lower extremities. The neuritis in the forearms produces first symmetrical finger-drop, with reaction of degeneration in the extensor communis, while the extensors of the wrist may at first be scarcely involved. The knee jerks, present at first, are likely to be lost after the first fortnight, and foot-drop and muscular wasting of the extensors may occur. In addition there is marked general weakness, so that the flexion of the hips becomes weak or completely paralysed, the arms cannot be raised at the shoulders and there is inability to sit up, from weakness of the back and abdominal muscles.

Family hæmatoporphyrinuria has been described by Barker and Estes [3] in America, and they ascribed the condition to an enterotoxin associated with great distension of the stomach and duodenum found at the autopsy. The action of sulphonal in producing hæmatoporphyrinuria varies greatly. Some samples of the drug have been known to produce hæmatoporphyrinuria with the

first dose. These cases are not serious and recover at once if the drug is stopped. In the majority of cases repeated daily doses of fifteen to thirty grains are necessary for several weeks or months to produce the disease. The appearance of port wine-coloured urine is then a very much more dangerous symptom, and is likely to be followed by increasing paralysis with the symptoms of neuritis already described, ending fatally.

Possibly the neuritis is not a direct result of the sulphonal upon the peripheral nerves, but may be due to an enterotoxin produced in some manner by the long-continued action of the drug. The acute hæmatoporphyrinuria followed by multiple neuritis, which sometimes occurs in the absence of any sulphonal or any other drug-taking, appears to be due to a similar enterotoxin, possibly produced in the stomach and duodenum, though the prime cause of such a disturbed metabolism is entirely unknown.

I have seen and studied two such cases,[23] one of them terminating fatally in a man who was apparently in perfect health, except for obstinate constipation until the onset of the disease with acute abdominal pains.

Indeed this case was mistaken at first for renal colic and then for an acute abdomen. It is these abdominal pains associated with obstinate constipation, insomnia and the early appearance of port wine-coloured urine, followed by pains in the limbs, with paræsthesiæ and muscular paralysis, that form the clinical picture of acute hæmatoporphyrinuric neuritis.

Polyneuritis Cranialis

Polyneuritis cranialis is a term which has been applied to cases in which several cranial nerves have been paralysed, usually on one side, but sometimes on both sides. The oculo-motor nerves usually escape, but any group from the fifth to the twelfth may be picked out. Thus facial palsy is common, and there may be nerve deafness, paralysis of the palate, vocal cord and wasting of the tongue. Occasionally such multiple cranial nerve palsies may be due to

secondary malignant growths in the meninges, and I have also seen a similar condition due to tuberculous caries at the base of the skull. Usually there is a history of chronic otorrhœa, though mastoid infection is the exception. Autopsies have shown these cases to be due to a basal infection in the posterior fossa, streptococcal in origin.

Gradual involvement of the oculo-motor nerves with severe chronic facial pain, with increasing anæsthesia of either the second or third division of the trigeminal nerve may be produced by growth in the naso-pharynx, such as naso-pharyngeal endothelioma, or malignant growths in the maxilla or in the sphenoid. The pain in these cases is chronic and wearing, varying at times in intensity, but never paroxysmal with intervals of total freedom from pain, such as are characteristic of tic douloureux.

Another important distinction between these two conditions is the presence of numbness and anæsthesia of parts of the trigeminal area, as there is never any anæsthesia in tic douloureux, unless after operation or alcohol injection treatment.

Polyneuritis in Hunger-œdema

During the later stages of the War a disease described as Hunger-œdema appeared in Austria and Germany, due to chronic starvation and malnutrition, probably associated with vitamin deficiency, as in beri-beri.

In 1919 Schlesinger described cases of polyneuritis as occurring in this disease.

GENERAL TREATMENT OF MULTIPLE NEURITIS

Rest in bed and warmth are essential preliminaries to treatment if severe pains are a feature of the disease, while massage, electricity and strychnine must not be employed so long as the pains continue, and alcohol must be entirely forbidden. Muscular tenderness in the calves, thighs, or forearms is also a common accompaniment

of the pains, and is a contra-indication for treatment by strychnine and electricity, though light massage may be bearable, and will then be of value. With the tenderness there is often in the first few weeks some contracture of the hamstrings, which may necessitate treatment by counter-extension with weights over pulleys, attached to padded leather anklets. These leather anklets should be made to lace up in front, and with a buckle firmly sewn on each side to which cords are fastened, and attached to a wooden stirrup below the foot, from the centre of which a cord runs over the pulley fixed by an iron rod to the foot of the bed.

The weights used will vary according to the amount of tenderness and what it is found that the patient will bear, three pounds usually being sufficient. Another method of treatment for flexor contracture at the knee is by the Macintyre double-inclined-plane splint, which has a screw attachment at the hinge under the knee by means of which the knee can very gradually be straightened by increasing the angle slightly daily. With this splint can be combined a footpiece fixed at a right angle when there is much foot-drop, in order to prevent the development of talipes equinus from contracture of the gastrocnemius.

Rarely muscular tenderness is combined with intense hyperæsthesia, so severe as to necessitate wrapping up the whole four limbs in thick cotton wool or Gamgee tissue. In time such hyperæsthesia and tenderness disappear, and then light massage can be begun, and later passive movements, daily galvanism of the paralysed muscles and injections of strychnine. The strychnine injections should be given in increasing doses, using the liq. strych. hydrochlor., beginning with five minims twice a day, and increasing the injection by one minim every other day, thus, five minims twice a day for the first two days, and then increasing the evening dose to six minims for the next two days, then for the fifth and sixth days both morning and evening the dose is six minims, and so on until nine minims is reached, which

dose should be given twice daily for three or four weeks. It should then be reduced again slightly and then stopped after a total course of six to seven weeks. This may be renewed, if necessary, after a fortnight's interval. Occasionally I use even larger doses of strychnine, as in cases where respiratory or cardiac failure is threatened in severe cases of multiple neuritis. As much as ten minims or one-tenth of a grain may be given with advantage every four hours for a day or two. Signs of over-dosage of strychnine by injection will be vertigo, and sometimes pain and stiffness in the muscles of the neck within five minutes of the injection. I have not, however, met with this symptom with a less dose than twelve minims of the liq. strych. Given thus hypodermically, there is no risk of any cumulative action of the drug, which may follow upon administration by the mouth. Indeed I once saw strychnine convulsions in a child of nine suffering from typhoid fever, who had been taking three minims of liq. strych. three times a day for a week.

With the gradual return of muscular power the question of how soon attempts at standing and walking are advisable must be approached. So long as there is any abnormality of the cardiac rhythm such as tachycardia, irregularity or bradycardia the patient had better remain strictly in bed, and not be allowed even to sit up. This involvement of the cardiac nerves is much more frequent in some forms of multiple neuritis than in others, particularly after diphtheria and beri-beri, though even in alcoholic neuritis a certain risk is also present. As soon as the heart's action shows no irritability on sitting up in bed, the patient may be moved out of bed to a comfortable chair for an hour or two a day.

Standing and walking are next attempted and support will at first be necessary, though soon the patient may be able to manage by pushing a chair in front about the room. Any tendency to foot-drop is more easily controlled as soon as the patient's weight is thrown on to the feet, but if the foot-drop has been excessive and untreated

by rectangular foot splints, and talipes equinus fully established by gastrocnemius contracture, then the patient will be practically unable to stand, and the question of bringing down the heel by lengthening the Achilles tendon will have to be considered. Simple section of the tendon Achillis is sometimes disastrous in its results, owing to non-union, but lengthening by Z-shaped incision may give very good results.

Neural Progressive Muscular Atrophy

In 1886 Tooth, in this country, and Charcot and Marie independently, observed a progressive form of muscular atrophy, occurring more commonly in males and commencing during the second decade of life or later.

Often familial and hereditary, in other instances the disease appears sporadically, and it affects symmetrically the muscles of the feet and legs, and later the intrinsic muscles of the hands and forearms. Club-foot and claw-hand commonly result, the knee and ankle jerks are lost, while sensory symptoms are present occasionally, though rarely. Autopsies have shown degeneration in the posterior and lateral columns of the cord, with anterior horn cell atrophy, and degeneration in the peripheral nerves—a chronic polyneuritis, hence the use of the term neural, or neuritic progressive muscular atrophy. I have seen a family in which the father and three out of five children were similar affected. All four had extreme pes cavus, with wasted legs and absent deep reflexes, but though the father and the two elder children, aged 11 and 7, had wasting of the lower part of the thighs, the youngest affected child, aged 4, had enormous thighs, the vasti interni especially being noticeably big.

There is often some difficulty in distinguishing this disease in the early stages from Friedreich's disease, the club foot and absent deep reflexes being common to both diseases, while the ages of the patients are similar, and both may have a family history of paralysis.

When the muscular wasting is pronounced this difficulty

will disappear, and the wasted clawed hands of neural muscular dystrophy are characteristic.

Progressive Hypertrophic Interstitial Polyneuritis

In 1893 Dejerine and Sottas described a similar familial muscular atrophy in juveniles, in which spinal cord changes of a like nature are found, but associated with chronic hypertrophic interstitial neuritis of the peripheral nerves. In addition to the muscular atrophy, severe shooting pains and anæsthesia are prominent symptoms, with ataxy, kypho-scoliosis, nystagmus and Argyll Robertson pupils, while many of the peripheral nerve trunks are noticeably enlarged. This form of the disease is much rarer than the neuritic form of Charcot–Marie–Tooth.

Hypertrophic Polyneuritis

Quite distinct from the above, signs of polyneuritis with palpable thickening of the nerve trunks due to interstitial inflammation may occur apart from leprosy, in which disease hypertrophic neuritis of many nerves is common. Leprosy should not be classed as multiple neuritis, as the nerves are damaged locally by bacterial invasion, not by a circulating toxin.

In 1921 Nattrass published a case of *recurrent hypertrophic neuritis* in a boy of 18, who had had three attacks of a similar nature, the two previous ones having occurred at the age of 4 years and again at 17, a year before the last attack. The paralysis was very complete of the limbs, and also of the trunk and neck muscles, with considerable muscular wasting, reaction of degeneration, and loss of the deep reflexes. There was muscular tenderness, and paræsthesiæ of the periphery of the limbs, but no cutaneous anæsthesia. The median, ulnar and external popliteal nerve trunks were much enlarged, nearly twice their normal size. Recovery was complete on each occasion after three or four months. Though there was no history of any similar family paralysis, Nattrass considers the case to be a modified and recurrent form of Dejerine and Sottas'

type of progressive hypertrophic interstitial neuritis of children. Beyond the fact of some hypertrophy of nerve trunks being present in Nattrass' case, the resemblance appears to end there, and I should consider it as an allied form to the cases of relapsing and recurrent polyneuritis previously published by Thomson and by Thomas and others.

Leprous Neuritis.

Though leprosy does not cause polyneuritis through the action of a circulating toxin, yet many nerves are commonly directly infected in this disease, giving rise to local hypertrophic neuritis of one or more nerve trunks, with corresponding paralysis of its motor and sensory supply. Hence leprosy has often been classified as a cause of multiple neuritis, and it will be convenient therefore to include it here. Occurring in two main forms, the nodular and the anæsthetic, the skin is mainly affected in the former, innumerable lepra bacilli being present in the skin and nodules. In the nervous form of the disease skin lesions are uncommon or absent, and lepra bacilli are not found in the skin, but patches of anæsthesia to tactile stimulation, and often also to painful stimuli and to pressure, with thickened nerve trunks, muscular wasting, glossy skin, perforating ulcer and other trophic lesions will be present, leading to spontaneous amputations and much deformity. The onset of the disease is often very gradual, and it may be entirely overlooked for several years, until patchy anæsthesia or local paralysis is noticed. Severe shooting and burning pains are common at times while the disease is advancing, and areas of cutaneous hyperæsthesia may be very troublesome.

In leprous involvement of the nerves a true ascending neuritis is produced, the bacilli invading and ascending the nerves from their cutaneous terminations, and in this manner a gradual ascending sensory and later motor paralysis of the periphery of the limbs may be produced. The two cranial nerves liable to be attacked in this manner

in leprosy are the fifth and seventh, trigeminal anæsthesia and motor facial palsy being not infrequent, which may be partial and bi-lateral. Thickening of the nerves from connective tissue infiltration is common, a form of hypertrophic neuritis, and such thickening, especially of the ulnar, peroneal or facial nerves, is a useful point for differential diagnosis from syringomyelia and from other forms of neuritis.

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CHAPTER III

NEURITIS OF CERVICAL PLEXUS

Cervical Plexus Neuritis

The most important branches of the cervical plexus are the phrenic nerve, derived chiefly from the fourth cervical nerve, and the great occipital nerve from the posterior division of the second cervical. The small occipital is also derived from the second cervical nerve, but from its anterior division, and it supplies the lower half of the back of the external ear and a strip of skin on the scalp behind the ear, between it and the area of the great occipital. The third cervical nerve supplies the integument of the upper part of the neck or collar area, and part of the external ear, the great auricular nerve being mainly derived from its anterior branch. The fourth cervical nerve supplies the skin outside the third cervical area as far as the point of the shoulder and downwards as far as the second rib.

The muscular supply of these nerves, with the exception of the phrenic, is not of much practical importance, with the exception of the posterior branches of the upper three cervical nerves which supply the complexus and the posterior rotators of the head, muscles which are concerned in the head movements of spasmodic torticollis, whose nerves require division in operations undertaken for the cure of wry-neck. Beyond neuro-fibrositis or muscular rheumatism affecting this area, the clinical importance of neuritis in the cervical plexus is mainly limited to occipital neuritis.

The Phrenic Nerve

The diaphragm, or midriff, is innervated by the phrenic nerve, which is made up of anterior branches from the

third, fourth, and fifth cervical nerves. Of these the branch from the fourth nerve is the largest and most important. Though placed midway in the trunk dividing the thorax from the abdomen, the diaphragm is a muscle which belongs developmentally to the neck region, as its nerve supply shows. Like the heart, which was originally placed high up in the neck above the larynx, as the winding of the recurrent laryngeal nerve around the aorta indicates, the diaphragm has also descended, carrying with it its cervical nerve supply.

In rare cases the left half of the diaphragm may be congenitally absent, in which case the lung is not developed on that side, and the stomach and intestines will be found high up in the thorax. This condition is liable to be mistaken for pleural effusion or pneumothorax, both of which mistakes I have seen made. An opaque meal followed by an X-ray photograph will at once demonstrate the true condition.

The phrenic nerve lies in front of the scalenus anticus muscle, and then passes in front of the subclavian artery and behind the vein to enter the thorax. It communicates with the nerve to the subclavius, crosses in front of the root of the lung and then lies between the pericardium and the mediastinal pleura, and then, reaching the diaphragm, it pierces this muscle to supply its under surface. On the left side the phrenic nerve crosses in front of the aortic arch before it reaches the root of the lung, but, unlike the recurrent laryngeal and sympathetic nerves, it is not liable to suffer from pressure of an aortic aneurysm.

The nuclear centre of the phrenic nerve in the spinal cord may be damaged by spinal cord disease, especially the chronic anterior poliomyelitis of progressive muscular atrophy. I have not seen it affected in acute poliomyelitis, though it will be involved in the terminal stage of an ascending myelitis. Transverse and destructive lesions of the spinal cord, as from fracture-dislocation of the spine, or acute myelitis below the level of the fifth cervical segment, will not cause any weakness of the

diaphragm, though all the intercostal and abdominal muscles will then be paralysed. In fact, owing to the paralysis of thoracic breathing, the diaphragmatic action in protruding the epigastrium and lower ribs during inspiration will be intensified. The roots of the phrenic nerve may be damaged by gummatous neuritis, by tuberculous caries of the spine or by new growth. The nerve may be severed in the neck by stab or gunshot wound, or it may become involved in a mediastinal tumour, as in a man of 58 I saw with total palsy of the inner cord of the brachial plexus and of the seventh cervical nerve, with sympathetic paralysis and diaphragmatic palsy on the same side, due to growth involving the upper lobe of the right lung. In such a case the unilateral action of the diaphragm on the damaged side is easy to demonstrate in comparison with the mobility of the lower ribs and epigastrium on the sound side. X-rays in such cases will show the dome of the diaphragm lying much higher than usual on the paralysed side.

Myositic Paralysis of the diaphragm may occur from general infiltration of the muscle with miliary tubercle, a condition I once saw in which a very expert clinician diagnosed perforative peritonitis owing to the complete loss of diaphragmatic action and the hyper-resonant note which had replaced the liver dulness. This was apparently due to the liver having fallen back and become covered by a distended colon.

Polyneuritis is a frequent cause of diaphragmatic paralysis, probably always symmetrical. This is a symptom not uncommonly seen in alcoholic neuritis and in the polyneuritis following diphtheria, lead poisoning, and many other causes of toxic polyneuritis. In diphtheritic neuritis the diaphragmatic palsy may alternate with intercostal paralysis.

Treatment of diaphragmatic paralysis is by strychnine—in young children one or two minims three times a day, while in adults six or seven or even ten minims of the liquor may be given hypodermically in acute and severe

cases. The phrenic nerve is sometimes irritated by chronic tuberculosis of the lung and pleura, causing the symptom of a dry, persistent cough. When there are unilateral signs of tuberculosis of the lower lobe in association with this form of cough, Morriston Davies advises section of the phrenic nerve on that side. This he does under local novocain anæsthesia where the nerve lies on the scalenus anticus in the neck. As a result of the immediate paralysis of that half of the diaphragm, the base of the lung is rested, being no longer expanded by the pulling action of the diaphragm on that side. The irritant cough disappears at once and the patient's condition may be much improved. No pain is felt on cutting the phrenic nerve under these conditions. This is probably due to the novocain anæsthesia, for it is certain that the phrenic is not a purely motor nerve, but contains sensory fibres conveying afferent impressions from the diaphragm. It is by this mechanism that the well-known "shoulder-tip" pain in sub-diaphragmatic inflammation is produced through the communications of the phrenic and the cervical plexus (see Visceral Neuralgias).

Hiccough

Hiccough or singultus is a well-known symptom in children, which may indicate nothing more than passing digestive disturbance, while in older people it is sometimes a sign of gravest portent. The phenomenon is due to a sudden spasmodic action of the diaphragm, but the mechanism is somewhat complex, as the diaphragmatic contraction is associated with sudden momentary closure of the glottis; this is not always caused by adductor spasm of the vocal cords such as occurs in laryngismus, but is due to the sudden negative pressure within the lung, produced by the diaphragm contraction pulling the vocal cords together, which will close from the down pressure of air from above, owing to the shape of the glottis. Hiccough may be a sign of grave danger in obstruction of the colon from carcinoma, after prostatectomy, or in cases of peritonitis following appendicitis or other infective processes.

Even the comparatively harmless operation of ligature of piles may produce hiccough, though it is not then of grave significance, unless it is allowed to continue for days, producing exhaustion.

I have found experimentally in cats that stimulation of the central end of the divided pelvic nerve causes a reflex violent contraction of the diaphragm, and it is probably this reflex that is at work in the production of hiccough in the pelvic disease above mentioned.

Persistent hiccough lasting for hours or even days may be a hysterical phenomenon, and I have seen this type occur in a man of 28 suffering with pseudo-hypertrophic muscular atrophy, the hiccough continuing for two days until arrested by strong general faradisation with the wire brush.

In another case of a man of 50, with a slow type of chronic anterior poliomyelitis and some symptoms of mental weakness, hiccough lasted for four days, causing considerable temporary exhaustion.

Acute lesions of the medulla may produce hiccough through a combination of irritation and partial paralysis of the motor vagus nuclei and the respiratory centre. I have seen troublesome hiccough in a man of 68, with acute symptoms of thrombosis of the left posterior inferior cerebellar artery, in whom, in addition to the usual syndrome of left trigeminal analgesia and partial tactile anæsthesia, with complete right-sided analgesia and thermanæsthesia of the body and limbs, excepting the face, there was obvious paralysis of the left half of the palate. There was slight conjugate deviation of the eyes to the left, with spontaneous nystagmus, intense vertigo on attempting to stand, and persistent hiccough.

Epidemic hiccough has been met with many times in association with outbreaks of encephalitis lethargica, and though not met with in severe or fatal cases, yet its occurrence in this form is considered as a manifestation of a slighter type of this disease.

The Treatment of Hiccough may be extremely difficult in cases of grave organic disease, and morphine, hyoscine or

inhalations of chloroform may become necessary. Traction on the tongue, gastric lavage or a hypodermic injection of one-tenth of a grain of apomorphine may arrest some stubborn cases, while less serious attacks may be terminated by the simple expedient of holding the breath, eating a piece of sugar, or by sniffing some strong scent, particularly snuff or pepper, in order to produce the reflex action of sneezing. These homely remedies have been known for over two thousand years, for Osler quotes Plato's Symposium, in which the physician Eryximachus recommended Aristophanes, who had hiccough from eating too much, either to hold his breath or to gargle with a little water, but if it still continued, "tickle your nose with something and sneeze, and if you sneeze once or twice, even the most violent hiccough is sure to go." The attack must have been of some severity, as it is recorded that the hiccough did not disappear until Aristophanes resorted to the sneezing.

CHAPTER IV

NEURITIS OF SHOULDER GIRDLE AND UPPER EXTREMITY

Nerves of the Upper Limb

The skin and muscles of the shoulder girdle and arm derive their nervous supply from one cranial nerve and eight spinal nerves, that is to say, the spinal accessory nerve and the third, fourth, fifth, sixth, seventh, and eighth cervical and first and second dorsal spinal nerves.

The spinal accessory nerve, though it is included amongst the cranial nerves and leaves the cranial cavity by the jugular foramen, is really a composite nerve, being made up of motor fibres derived in part from the nucleus ambiguus in the medulla, and of motor fibres derived from the ventro-lateral horn of the cervical segments of the spinal cord as low down as the sixth cervical. These fibres, instead of issuing from the spinal foramina like the rest of the anterior root fibres, turn upwards and enter the cranium through the foramen magnum, and join with the motor fibres from the nucleus ambiguus.

These latter fibres again separate from the spinal portion at the jugular foramen and become part of the vagus system, supplying the palate and vocal cord with motor innervation. The spinal portion is continued as the spinal accessory nerve, and pierces the sterno-mastoid muscle, which it supplies with branches as it runs obliquely through the muscle, leaving it in the middle of its posterior border. From there it crosses the posterior triangle and enters the trapezius muscle, which it supplies.

Trapezius Palsy

This muscle together with its fellow on the opposite side forms a quadrilateral diamond-shaped sheet of muscle

attached to the occipital protuberance above in the middle line and laterally to the spine and acromion process of the scapula, and to the spines of the lower six dorsal vertebræ. Its shape is supposed to resemble a monk's cowl, hence the

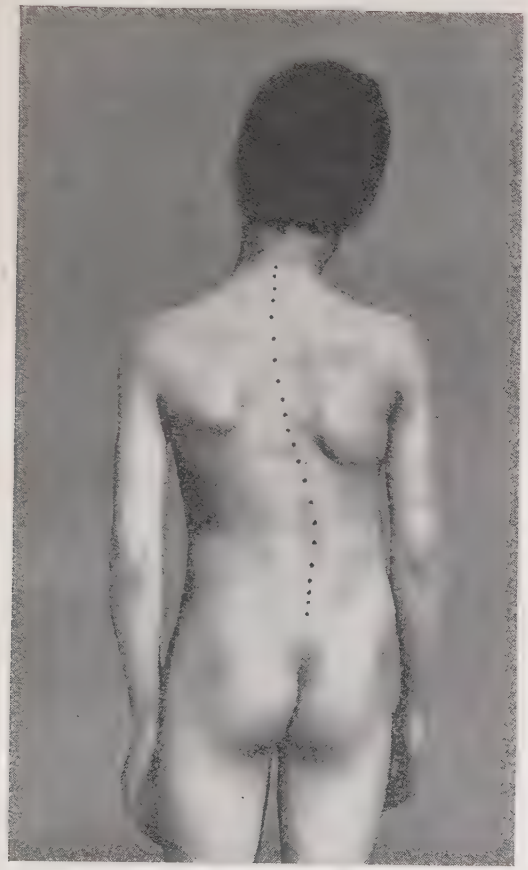

Fig. 1.—Paralysis of left trapezius.

name musculus cucullaris. By its action the scapula and arm are retracted and drawn inwards and downwards. The upper portion, sometimes known as the respiratory portion, is thicker than the rest, and by its action helps to raise the scapula, and also helps to retract the head and

rotate it towards the opposite side. The middle and lower fibres, however, pull the scapula downwards and inwards. Consequently in cases of paralysis of the trapezius the scapula slips upwards by the unopposed pull of the levator anguli scapulæ, and the whole shoulder girdle slips outwards at the same time, so that its vertebral border now lies from one and a half to two inches further away from the dorsal spines, and its inferior angle higher than that of the scapula of the opposite side (see Fig. 1). The inferior angle is also slightly everted, giving an appearance of what is called " winged " scapula. Owing to the wasting of the thick muscular upper portion of the trapezius, the shoulder now appears squarer than its fellow, and the clavicle can be seen from behind, whereas when viewed from the front the upper angle of the scapula is seen prominently, almost overhanging the clavicle, and the attachment of the levator anguli scapulæ stands out distinctly (see Fig. 2).

Uni-lateral paralysis of the trapezius does not produce great disability in the use of the arm, but interferes with the power of carrying weights upon the shoulder, and with movements especially involving the shoulder in outdoor games and sports, such as swimming, tennis and cricket.

Causes of Trapezius Paralysis

This may be congenital in the form known as Sprengel shoulder, or it may share in the atrophy of muscles in different forms of idiopathic muscular atrophy, or myopathy, especially Erb's juvenile type, the pseudo-hypertrophic and Landouzy-Dejerine forms. In these types there is no injury or neuritis of the spinal accessory.

Trapezius paralysis may also occur from spinal cord disease in syringomyelia, and in the spinal form of progressive muscular atrophy, in either that form occurring in adults after the age of 40 or in the infantile form described by Werdnig and Hoffmann. The upper or respiratory portion is usually only slightly involved in progressive muscular atrophy, and dropping forward of the head and chin with inability to hold the head up does not occur

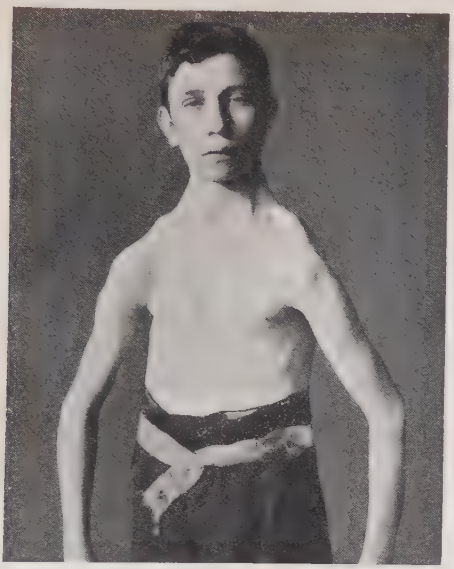

FIG. 2.

FIG. 3.

Bilateral trapezius paralysis, following operations on glands in the neck.

unless the complexus and deeper muscles are involved in the paralysis. In these cases the lesion is, of course, in the anterior and lateral horn cells in the cervical cord.

The spinal accessory nerve is liable to injury in the neck in operations for the removal of glands and growths, and I have seen bi-lateral total trapezius palsy in a boy from this cause (see Figs. 2 and 3). If the diseased glands are removed below the angle of the jaw, then the spinal accessory nerve may be destroyed before it enters the sterno-mastoid, in which case both that muscle and the trapezius will be permanently paralysed and wasted. In the operation, too, of facio-accessory anastomosis for total persistent facial paralysis, the spinal accessory is liable to be damaged, and I have seen total sterno-mastoid and trapezius paralysis from this cause without any benefit to the facial nerve. It is usual, however, now to use the hypo-glossal nerve rather than the spinal accessory for this anastomosis operation. The nerve is also liable to be damaged in the operation for spasmodic torticollis, but this may easily be avoided by splitting the sterno-mastoid obliquely along the course of the nerve as it pierces the muscle, and lifting out the nerve after cutting off the branches which it gives to the sterno-mastoid.

If the nerve is damaged by glands or tumour between the jugular foramen and the anterior border of the sterno-mastoid, both trapezius and sterno-mastoid muscles will be paralysed together, but if the nerve is damaged in the posterior triangle after leaving the posterior border of the sterno-mastoid, then the trapezius suffers alone. Usually practically the whole muscle wastes away in total spinal accessory paralysis, although the muscle has an additional supply from the third and fourth cervical nerves.

It is possible that these spinal nerves supply sensory more than motor innervation to the muscle.

Occasionally as a result of neuritis the middle and lower portions only of the trapezius are paralysed, causing partial winging of the scapula (see Fig. 4).

This position is brought about by the scapula slipping

forwards and outwards, while the action of the rhomboids in pulling the inferior angle inwards exaggerates the tilting caused by the forward and outward movement of the acromion. This partial winging of the scapula from paralysis of the middle and lower trapezius fibres is very

Fig. 4. Fig. 5.

Paralysis of middle and lower portions of right trapezius. Fig. 4 illustrates the partial winging of the scapula. Fig. 5 shows the disappearance of the winging when the undamaged serratus magnus is thrown into action.

commonly mistaken for serratus magnus palsy, due to neuritis of the long or posterior thoracic nerve. This error may be avoided by testing the action of the serratus in pulling forward the scapula, as in reaching forward with the arm, when the winged appearance vanishes if the serratus is acting normally (see Fig. 5).

Neuritis of the spinal accessory is rarely very painful,

though a certain amount of aching of the shoulder may occur with over-use of the arm and shoulder.

Sprengel Shoulder.—This term is applied to varieties of congenital malformation of the trapezius and scapula. The muscle may be congenitally absent, or it may be shrunken and fibrosed, possibly from intra-uterine damage, the scapula being more or less fixed and usually smaller than its fellow.

There is sometimes bony malformation in which a bridge of bone extends from the scapula to the vertebral column, either from the upper angle of the scapula, or from its

Fig. 6.—Hysterical contracture of head and shoulder.

spine, thus fixing the scapula. X-ray examination may be necessary to distinguish between these forms.

Spasm of the trapezius may be either constant or intermittent. Constant or tonic spasm of the muscle may be hysterical, the shoulder being drawn up and the head drawn down to meet it. Much commoner is irregular or clonic spasm of the muscle. This may occur as a shoulder-shrugging movement in habit spasm or tic, or it may be associated with clonic spasm of the sterno-mastoid of the same side, and the posterior rotators of the head on the opposite side in spasmodic torticollis (wry-neck).

Posterior Scapular Nerve

This nerve, the dorsalis scapulæ, is sometimes called the nerve of the rhomboids, and is a posterior branch from the

ventral division of the fifth cervical nerve. It assists the third and fourth cervical nerves in supplying the levator anguli scapulæ, and then innervates the rhomboid muscles, and also gives a branch to join the posterior thoracic nerve for the supply of the upper portion of the serratus magnus. The posterior scapular nerve is given off close to the spinal

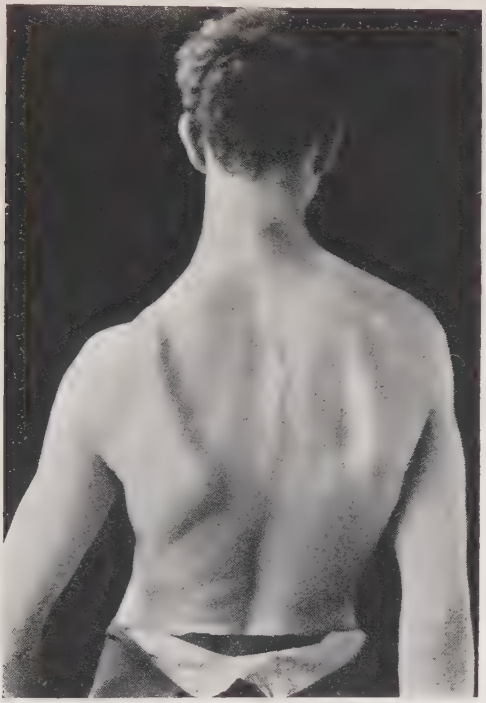

Fig. 7.—Paralysis of left trapezius, levator anguli scapulæ and rhomboids.

foramen, and is rarely damaged by direct injury. I have seen it paralysed from a gunshot wound and also by violent sudden muscular action in raising the shoulder; as a result the scapula hangs somewhat lower with its inferior angle everted, the rhomboid paralysis being well demonstrated in the action of pressing down the elbow against resistance, when the inferior angle of the scapula slips outwards instead of being held firm by the rhomboids.

In post-influenzal brachial neuritis I have seen the rhomboids paralysed without the levator anguli scapulæ, in association with the serratus magnus, latissimus and pectoralis major.

Long Thoracic Nerve

The long thoracic or external respiratory nerve of Bell, like the dorsalis scapulæ, is made up by posterior

Fig. 8.—Winged scapula due to paralysis of right serratus magnus in a case of rheumatic polyneuritis.

branches from the ventral divisions of the fifth, sixth and seventh cervical nerves. The branch from the sixth cervical is the largest of the three, and arises from the back of the sixth cervical nerve further away from the spinal foramen than the other two branches; it pierces the scalenus medius and crosses the posterior triangle to supply the serratus magnus.

Serratus magnus paralysis causes weakness of the forward

movement of the shoulder, with inability to raise the arm above the horizontal. When the arm is held straight in front, the weight of the arm makes the scapula fall backwards, its vertebral border protruding greatly as " winged " scapula.

Owing to the paralysis of the middle and lower fibres of the serratus, the scapula cannot be rotated forwards, and

Fig. 9.—Paralysis of both trapezii and serratus magnus muscles in a case of Erb's juvenile muscular dystrophy.

consequently the arm cannot be raised straight above the head, as the deltoid muscle raises the arm only to the horizontal position, further movement of the arm to the vertical position being produced by the serratus swinging the lower border of the scapula forwards.

In its spinal supply the serratus magnus is associated with the latissimus dorsi, triceps and sternal portion of the pectoral muscles, so that in lesions of the sixth cervical level of the cord this group of muscles is apt to be paralysed,

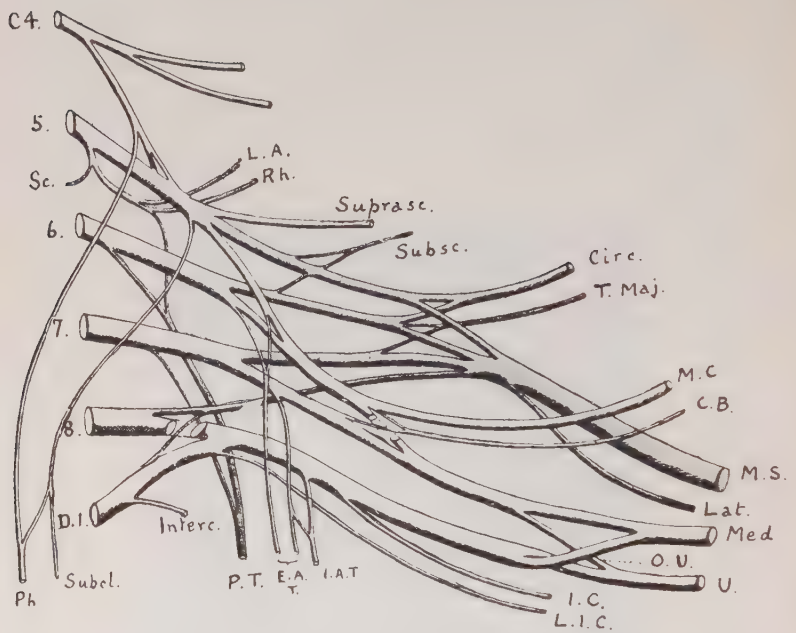

FIG. 10.—BRACHIAL PLEXUS (Prefixed type).

The connective tissue sheaths of the nerves are all dissected off, and thus the posterior and outer cords are seen resolved into their component parts. The branch from 5–6 C to the outer cord has been divided in order to show the origin of the nerve to the coraco-brachialis; the branch from 8 C to the inner cord has also been similarly divided to show the posterior branch of 1 D. Note the large size of the outer head of the ulnar, piercing directly through the inner head of the median. Note also that the inner head of the median is given off from the lower and anterior portion of the inner cord, and passes in front of the ulnar, which is given off from the posterior bundles of the inner cord. The posterior cord (shaded) lies behind and slightly above the outer and inner cords.

C 4, 5, 6, 7, 8, D 1. Anterior divisions of the fourth, fifth, sixth, seventh, and eighth cervical nerves and the first dorsal nerve. Sc., Scalene. Ph., Phrenic, to diaphragm. Subcl., Subclavian. Interc., Intercosto-humeral. L.A., Levator anguli scapulæ. Rh., Rhomboids. Suprasc., Supra-scapular, to supra- and infra-spinatus. Subsc., Sub-scapular. Circ., Circumflex, to deltoid. T. maj., Teres major. M.C., Musculo-cutaneous, to biceps and brachialis anticus. C.B., Coraco-brachialis. M.S., Musculo-spiral, to triceps and extensors in forearm. Lat., Latissimus dorsi. Med., Median. O.U., Outer head of Ulnar. U., Ulnar. I.C., Internal Cutaneous. L.I.C., Lesser internal cutaneous. I.A.T., Internal anterior thoracic to pectorals. E.A.T., External anterior thoracic to pectorals. P.T., Posterior or long thoracic, to serratus magnus.

as in acute poliomyelitis. The nerve may be damaged by violent action of the scalenus medius, which I have seen as a result of using the side stroke in a swimming race. The nerve may also be damaged from pressure in the posterior triangle of the neck by the carrying of weights on

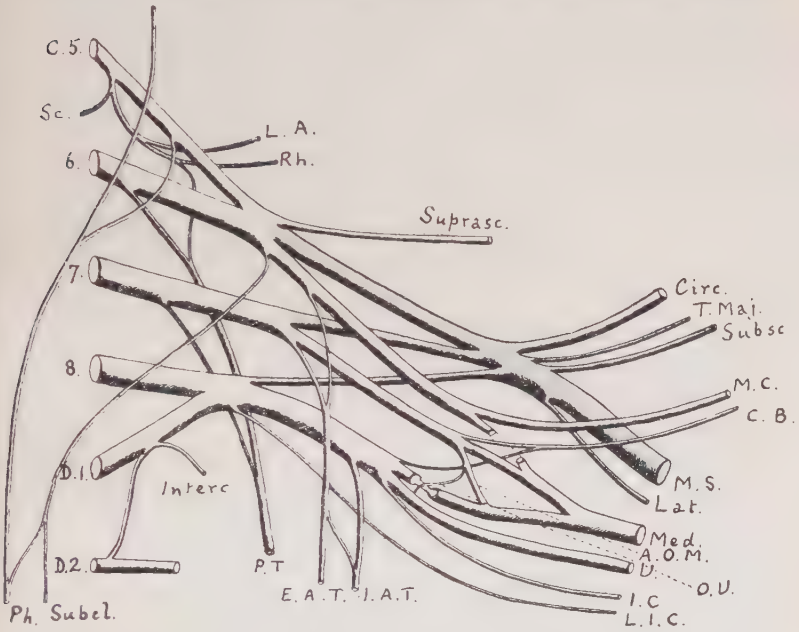

FIG. 11.—BRACHIAL PLEXUS (Postfixed type).

In this case the nerve sheaths have not been dissected off the posterior cord, but the outer cord has been dissected to show the origin of the nerve to the coraco-brachialis from 7–8 C. Note the small size of 5 C, and also the fine recurrent branch which leaves the accessory outer head of the median, and perforates the inner head of the median to form the outer head of the ulnar. Note the ascending branch from D 2 to join D 1.

A.O.M. ..... Accessory outer head of median.

Other lettering as Fig. 10.

the shoulder, and it may also suffer as the result of an acute neuritis from chill, influenza, or after paratyphoid. In these latter cases the paralysis is preceded by considerable pain around the back of the shoulder and neck. This muscle is often paralysed, with other muscles of the shoulder girdle, such as the trapezius, in the muscular dystrophies.

Brachial Plexus Paralysis

Though the two preceding nerves, the posterior scapular and long thoracic, are posterior branches of the brachial plexus, yet they are practically never involved in lesions of the brachial plexus as a whole, owing to their being given off from the ventral divisions of the spinal nerves almost immediately after their exit from the spinal foramina. The brachial plexus is made up of the ventral or anterior divisions of the fifth, sixth, seventh and eighth cervical and first dorsal nerves, together with a small branch from the fourth cervical and another from the second dorsal. The brachial plexus may be shifted up or down, so to speak, about half a spinal segment in its origin, so that the plexus may be called high, or prefixed plexus, and low, or post-fixed. In the former, the fifth cervical trunk is large, and there is a quite definite branch from the fourth cervical to join it. In such a case there may be no branch from the second dorsal to join the first dorsal.

In the prefixed plexus also there is a fair-sized branch, which I have called the outer head of the ulnar nerve, passing from the outer cord and piercing the inner head of the median to join the ulnar nerve, thus carrying innervation from the seventh cervical segment into the ulnar nerve, probably for the flexor carpi ulnaris. In the low or post-fixed plexus there may be no branch from the fourth cervical to join the fifth, which is then much smaller than the sixth, perhaps only one-quarter of its size. The branch from the outer cord to the ulnar is then very fine and only to be found with difficulty, the nerve to the coraco-brachialis is derived from the seventh and eighth nerves, instead of from the sixth and seventh, and the ascending branch from the second dorsal nerve to join the plexus will be quite distinct. This latter carries the sensory fibres to the intercosto-humeral nerve from the second dorsal segment. Owing to this variability of high or low set plexus, muscular innervation will vary in different people to a certain extent as regards spinal segmentation.

Roughly the distribution of the spinal segmentation is as follows :

5th Cervical.—Deltoid, biceps, brachialis anticus, supra- and infra-spinatus, supinator longus.

6th Cervical.—Serratus magnus, latissimus dorsi, triceps, lower pectoral.

7th Cervical.—Extensors in the forearm.

8th Cervical.—Flexors in the forearm.

1st Dorsal.—Intrinsic musculature of the hand.

A notable exception to this grouping is the radial extensors of the wrist, which are supplied by the fifth and sixth cervical nerves.

The brachial plexus may suffer as a result of direct violence from stab or gunshot wound, or by violent wrenching of the shoulder or neck, or again it may be damaged by peri-neuritic inflammation of rheumatic type, known as brachial neuritis. Growths, too, in the neighbourhood of the scapula and posterior triangle may involve one or more trunks of the plexus and occasionally the inner and posterior cords of the plexus may suffer from spread of inflammation from the pleura, as in tubercular pleurisy.

Erb-Duchenne Paralysis.—Erb described in adults and Duchenne in new-born infants paralysis of the fifth and sixth cervical nerves from injury, causing paralysis of the deltoid, spinati, and flexors of the forearm, including the supinator longus. As a result of this paralysis the arm is held closely to the side and extended at the elbow, and is rotated inwards by the sub-scapularis. Partial paralysis of this type may be met with in which the deltoid and spinati muscles alone suffer, the flexors of the elbow escaping almost entirely.

This condition may be simulated by the arthritic atrophy met with in tuberculosis or in severe osteo-arthritis of the shoulder joint, and the same muscles may occasionally be paralysed alone in acute poliomyelitis.

The fifth cervical, being the upper nerve of the plexus, is the most obliquely placed and is consequently the most

liable to injury from downward traction of the arm, especially when the head is wrenched to the other side. Thus traction of the arm by finger or hook in cases of breech presentation may produce this type of paralysis in new-born children.

In adults, it is usually the result of a fall on the point of

Fig. 12.—Erb's paralysis, left arm.

the shoulder from a height of two or three feet, as out of a cart on to the hard road, the head being violently pressed or forced to one side, thus suddenly stretching and perhaps rupturing the uppermost nerve of the plexus.

This type of paralysis, or Erb's palsy, as it is often called, is usually ascribed to a lesion of the fifth and sixth cervical nerves, where they join together at the outer border of the scalenus medius, and indeed scar tissue may be found

involving the combined trunk at this point. If, however, during an operation on the nerves for the purpose of removing such scar tissue or performing nerve anastomosis, the fifth and sixth cervical nerves be carefully isolated by a vulcanite spatula, and separately stimulated with a weak

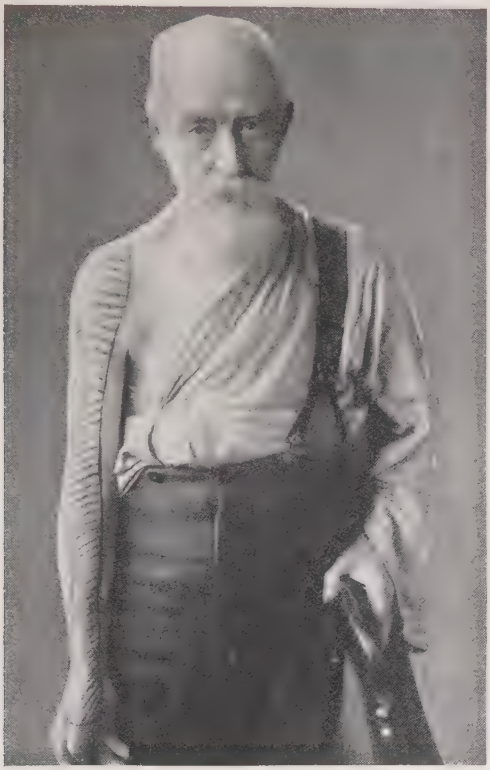

FIG. 13.—A case of Erb's palsy due to acute neuritis, with extensive herpetic eruption over the cutaneous area of the fifth cervical root, outlined and shaded in black.

current from a pointed sterilised electrode, I have generally found that strong contractions in the arm result from stimulating the sixth cervical nerve, while none may be obtained on stimulating the fifth. Indeed the fifth cervical nerve fibres may be completely ruptured within the sheath.

Nerve suture and nerve anastomosis have been done

for this condition with but indifferent success as a rule, though Kennedy has reported good results in children by cutting out the area of scar tissue with the damaged nerves and suturing the fifth and sixth nerves to the cut ends of the nerve trunks below.

Though in many cases of Erb's palsy there is complete flaccid paralysis of abduction of the shoulder and of flexion

Fig. 14.—Anæsthesia in a case of gunshot injury of the left fifth and sixth cervical nerves.

b. Loss to pin-prick and to light touch.
c. Diminished perception of light touch and pin-prick.

of the forearm, with atrophy of those muscles, yet there may be no detectable anæsthesia anywhere on the arm. In a few cases I have seen numbness and partial anæsthesia of the thumb and index finger, which is apparently due to involvement of the sixth cervical nerve, but when the stress of the lesion falls upon the fifth cervical nerve only, as I believe it does in the majority of cases, then there is no anæsthesia to be detected. This, I think, is due to the fact of only one nerve root being involved, the overlapping of neighbouring roots making up for the loss of one root area.

Moreover, the fifth cervical nerve has a larger proportion of motor fibres in it than any of the other nerves that form the brachial plexus.

This is due to the large size of the muscles supplied by it, and to the fact that the strip of skin from the outer side of the shoulder down the arm to the wrist which is supplied by the fifth cervical nerve has a very low cutaneous sensibility compared with the fingers and hand, which are supplied by the sixth, seventh, and eighth cervical nerves. In them tactile discrimination reaches its highest point of development, requiring much richer sensory nerve supply than on the skin of the arm and forearm.

The whole brachial plexus may be torn from its origin at the foramina by great violence, as when an arm is caught in revolving machinery. I have seen this result in a boy who when riding a bicycle was crushed between two passing tram-cars. Total flaccid paralysis of the arm resulted, with wasting and degeneration of the muscles, and complete anæsthesia of the hand and forearm and lower part of the arm. In such an accident the first dorsal nerve is liable to be torn close to the foramen, so that the branch going to the cervical sympathetic is involved, with resulting slight ptosis on that side and contracted pupil. No pain as a rule follows from this severe nerve injury.

The damage may be limited to the lower part of the plexus only, the eighth cervical and first dorsal nerves, which join to form the inner cord, being liable to be damaged by an upward and backward pull on the arm.

This condition is sometimes known as Klumpke paralysis, and causes wasting of the intrinsic muscles of the hand and paresis of the flexors of the fingers, with anæsthesia along the inner side of the forearm, hand and little finger.

Rib Pressure.—The first dorsal nerve not infrequently suffers from pressure owing to a cervical rib, in which case the nerve has to rise higher into the neck, in order to reach and join the eighth cervical nerve.

The symptoms produced are those of pain referred along the inner side of the arm and hand, and in the neck, with

gradual atrophy of the thenar eminence and interossei muscles. Vasomotor phenomena too are common, swelling and blueness of the hand being met with occasionally. In some cases the bony cervical rib itself is short, but is continued as a fibrous band to join the first rib, and such a fibrous cord will not be evident in an X-ray photograph. Moreover, X-rays may show no abnormality whatever, and yet all the phenomena of rib pressure may be present, the first dorsal nerve being compressed as it rises over the first rib. Skiagrams therefore are not conclusive in the differential diagnosis of cases whose symptoms may suggest rib pressure. The symptoms of this condition are not met with in childhood, but are liable to appear after puberty is reached, and are considerably commoner in the female than in the male, perhaps owing to the more sloping shoulders in the former sex. Rarely will signs of rib pressure commence after the age of 30. Operations for the relief of these symptoms have met with only partial success, but occasionally very good results have been observed after the removal of a cervical rib or of its fibrous band, or of the middle portion of the first rib, which occasionally acts in like manner.

Circumflex and Supra-scapular Nerve Injuries

The circumflex nerve may suffer occasionally from pressure, as from a crutch or from elevation of the arms during sleep, causing paralysis of the deltoid. The cutaneous branch may escape, but generally there will be light anæsthesia to touch and prick over the outer and upper part of the arm.

The deltoid is a most important muscle in the upper extremity, practically no other muscle giving it any assistance in abducting the arm. The clavicular pectoral has been stated to have an abductor action, but this is an error, this muscle contracting only when the arm is held straight forwards in adduction, and its action is adductor and prevents the normal action of the deltoid of abduction from the middle line.

The infra-spinatus rotates the arm outwards, and this movement will suffer in supra-scapular neuritis. This nerve may rarely be damaged by carrying weights on the shoulder, but is most commonly damaged together with the circumflex in dislocations of the shoulder.

Dislocation of the Shoulder

Downward dislocations of the shoulder, by forcing the head of the humerus into the axilla, not infrequently cause injuries to various nerves as they enter the arm from the brachial plexus. The most common nerves to be damaged are the circumflex and the branch of the supra-scapular to the infra-spinatus muscle. By the downward dislocation of the head of the bone the circumflex nerve is stretched as it winds round the head of the bone to enter the under surface of the deltoid muscle, while the infra-spinatus is paralysed on account of the muscle being torn away from its nerve as the latter winds under the acromion process to enter the under surface of the muscle.

Owing to the pain resulting from the dislocation, and to the arm being kept bandaged in a fixed position, paralysis of these two muscles is often overlooked after this accident. If there is complete inability to abduct the arm when passive movements of the joint are first performed, the electrical reactions of the muscles should be examined after seven days have elapsed since the accident, and reaction of degeneration, if found, will indicate lesion of the circumflex nerve, and affects the prognosis as regards recovery, as ability to abduct the arm is not likely to return for six months or more. Paralysis of the infra-spinatus will be made evident by rapid wasting of the muscle below the spine of the scapula, though any loss of power due to its paralysis is hardly likely to be noticed at first. Its action is to rotate the arm outwards, and it is especially used in the act of writing. This deltoid and infra-spinatus paralysis following dislocated shoulder somewhat resembles a partial Erb's palsy, or a very localised poliomyelitis, but may be distinguished by the presence of anæsthesia on the outer

and upper arm, due to the circumflex injury. This anæsthesia is not present in Erb's palsy.

Usually the biceps is involved more or less completely in Erb's palsy, but then the supinator longus will also be involved. The biceps may also suffer from dislocation of the shoulder through pressure on the musculo-cutaneous nerve, but the supinator longus will not be affected, unless there is also paralysis of the whole musculo-spiral distribution.

Occasionally, the nerve injury from pressure in the axilla, after dislocation of the shoulder, is very severe and widespread, and there may be total paralysis of the whole upper limb from damage to the various nerve trunks, circumflex, musculo-spiral, musculo-cutaneous, median and ulnar.

In such a case there will be total anæsthesia to touch, prick and pressure on the whole hand and forearm. I have seen a case in which only the musculo-spiral nerve escaped, though in this case the latissimus was paralysed through involvement of the long subscapular nerve. The pectoral muscles never suffer from this injury, nor the serratus magnus, as the anterior and posterior thoracic nerves always escape. Even in rupture of the whole plexus the serratus magnus generally escapes, owing to the origin of the long thoracic from the posterior aspect of the fifth, sixth and seventh cervical nerves close to the intervertebral foramina.

Next to the circumflex and supra-scapular nerves the musculo-spiral is the commonest nerve to be damaged by the pressure of the head of the bone in the axilla, causing paralysis of the triceps and of all the extensor muscles of the wrist and fingers. The median and the ulnar nerves may also suffer in like manner. Usually the pain from these nerve injuries is not very severe and lasting, but I have seen most intense and persistent causalgia of the median nerve distribution follow immediately after dislocation of the shoulder in an old lady of 70.

The whole brachial plexus may be compressed between the clavicle and first rib during sleep or under an anæsthetic,

and flaccid paralysis of the whole limb from sleep palsy has been twice recorded.

Acute Brachial Neuritis

Acute perineuritis of the nerve trunks of the brachial plexus is comparable to acute sciatica in the lower extremity. Often of rheumatic or gouty origin, it may arise from a chill or from overstrain of the muscles of the arm, or may follow acute infections like influenza or typhoid.

Not infrequently brachial neuritis seems to occur without any adequate cause, though it is usually preceded by aching pains in the muscles at the back of the neck and head, with stiffness of the neck.

This is probably a fibrositis, which may spread forward to the nerve sheaths of the brachial plexus, setting up acute brachial neuritis, just as lumbago frequently precedes sciatica. Fibrositis again may result from many forms of infection, such as pyorrhœa, dental abscess, colitis and chronic appendicitis, and therefore it is possible that the extraction of a tooth may be necessary to cure the pain in the arm, just as the same treatment may undoubtedly relieve an obstinate lumbago. The pain of brachial neuritis spreads from the shoulder and neck within a day or two down to the tips of the fingers, the two middle fingers usually being the most painful. At first pins and needles sensations only, the pain becomes more and more severe, till within a week it may be most excruciating, abolishing sleep and comparable to knives cutting or to rats gnawing the flesh off the bones. There may be some swelling of the hand and arm, and movements aggravate the pain, with hyperæsthesia of the skin and intense tenderness of the whole limb, so that it may be necessary to wrap the limb up in thick wadded quilts or cotton wool and rest it on a down pillow. Fixation of the arm to the side by splint or bandage cannot, however, be borne; change of posture becomes an absolute necessity for the patient, even though the suffering may be temporarily increased thereby. The pain continues day and night, though exceptions occur, and

sleep has to be secured by the aid of hypnotics combined with analgesic drugs. The natural salicylate of soda, or aspirin combined with phenazone or pyramidon and medinal, with a little heroin, will usually abate the intensity of the pain, and allow sleep to follow. Local applications to the limb of glycerine and belladonna or poppy fomentations sometimes are of service, though electricity I have generally found aggravates the pain. Faradisation must, of course, not be used, but ionisation carefully applied through a sponge electrode over the posterior triangle, with the hand in water and a constant current passed through the limb of ten to twelve milliamperes, is a rational treatment to try, and is certainly sometimes very valuable in the corresponding condition of sciatica. Radiant heat and diathermy may also be tried, the former applied by a reflector containing one or two large lamps. The leucodescent lamp and blue light have also their advocates.

The duration of the severe pain varies from a few days to a month, but it rarely drags on for months or even years as sciatica quite frequently does. Occasionally severe joint fixation follows brachial neuritis, the commonest being fixed shoulder from adhesions, due to the immobilisation of the joint for several weeks.

It is well to warn the patient at the commencement of the trouble that stiffness of the shoulder will surely result if the pain continues for several weeks. This stiffness may be overcome by passive movements and massage as a rule, but sometimes requires wrenching under gas anæsthesia.

The elbow joint is rarely affected, but periarticular adhesions of the inter-phalangeal joints of the fingers may be very troublesome. Other trophic evidence of the neuritis will be wasting of the finger pads and striation and incurving of the nails and glossy skin. In rare cases even subluxation of the inter-phalangeal joints is met with. I have seen this with glossy skin resulting from severe brachial neuritis secondary to tubercular pleurisy and consolidation of the upper lobe of the lung on that side.

Adhesions in the Shoulder Joint

Pains around the shoulder and upper arm, often referred to the middle of the outer side where the deltoid is inserted into the humerus, with stiffness of the shoulder joint and inability to raise the arm properly, are a common sequel of a sub-acute rheumatic inflammation of the shoulder joint, or of slight injuries in this region. A considerable number of such cases have been wrongly diagnosed as "neuritis," on account of the pain and consequent disability. Blows, as from a fall on to the shoulder, or a sudden wrench of the shoulder, as by the jerk of a starting bus or tramcar, while yet the back rail of the car is held in the hand, may set up an acute synovitis of the shoulder joint, pains appearing two or three days after the slight injury, which may have been overlooked and half forgotten. Owing to the pain the shoulder is kept more or less stiff, and adhesions soon result, so that when all active inflammation has passed away the shoulder joint is more or less fixed by peri-articular adhesions, which give rise to constant pains with every little movement of the shoulder. Gradually a certain amount of atrophy of the deltoid and infra-spinatus muscles develops, the so-called arthritic atrophy, which affects chiefly the abductors and extensor muscles of a painful and inflamed joint.

This atrophy is usually ascribed to disuse, but it is sometimes so extreme, as in tubercular joints, as to resemble poliomyelitis or severe nerve injury.

Before commencing treatment of such a fixed shoulder it is advisable to have an X-ray photograph of the joint, in order to exclude osteo-arthritis and lesions involving the bone and cartilage. The aim of treatment is to re-establish mobility of the joint, by stretching, rupturing, or otherwise causing disappearance of the adhesions. Long-continued ionisation and massage of the shoulder usually fail to make any impression; the same may be said of radiant heat and diathermy and other forms of local

treatment. There are only two practical methods of dealing with the condition. One way is to persist steadily with passive movements, massage and exercises so as gradually to stretch the adhesions.

This is likely to be a long process, and often fails on account of the inability of the patient to relax. The scapula not being fixed to the vertebral column but only slung by muscular attachments, develops an increased mobility when the shoulder joint is fixed, and all attempted movements of the joint are apt to result in movements of the scapula and arm as a whole without the joint itself moving. In such cases the joint should be thoroughly moved and the adhesions broken down under nitrous oxide anæsthesia. Details of this little operation are worth some attention. As a preliminary it is a useful procedure to poultice the shoulder joint continuously for several days, and the masseur who is to carry on the passive movements of the joint daily should be present at the operation, in order to satisfy himself that the adhesions have been broken, and that all resistance to free movements of the joint afterwards is due to muscular spasm alone.

When the patient has been put under the anæsthetic the scapula should be firmly gripped in one hand and the arm seized by the other hand above the elbow, and the joint then moved gradually with increasing movements in all directions, especially full abduction and outward rotation. As this is done adhesions can be heard and felt to break like crackling twigs; the arm is firmly pulled up straight over the head, and, in order to provide for this movement, the patient should lie slightly diagonally across the couch with only one pillow under the head. The anæsthetic is now stopped and the patient is allowed to come round while the arm is held straight above the head. The operator's other fist is firmly pressed into the axilla in order to prevent dislocation of the shoulder joint, which might easily occur when the arm is brought downwards. The patient is now made to realise the extended position of the arm, which is then brought

down to the side and immediately raised again straight above the head.

This causes a little pain but is of value in demonstrating its possibility to the patient. The arm is then brought down again, a fresh poultice applied to the shoulder and a quarter of a grain of morphine given.

The same evening the masseur should move the arm fully again, and this should be repeated twice daily, while the patient is also encouraged to practise raising the arm, standing flat against a wall while the toes and chest are touching the wall; the aim should be each day for a higher point to be reached by the fingers on the wall until it approximates to that possible with the undamaged arm.

A similar treatment for the hip joint may occasionally be cautiously employed with advantage, but the utmost care must be taken with the hip joint not to apply much force owing to the danger of fracturing the femur, as the leverage here is very great, and the pelvis, unlike the scapula, is fixed to the vertebral column.

Circumflex Neuritis

This nerve supplies the deltoid and teres minor muscles, and also a flap of skin over the shoulder joint down to the middle of the outer side of the arm. Injury of this nerve by dislocation of the shoulder has already been referred to. It may also be involved in fracture of the neck of the humerus and in gunshot and stab wounds of the shoulder.

Neuritis of the circumflex is not uncommon after paratyphoid, and it may be specially prominent in some forms of multiple neuritis, such as the upper arm type of lead palsy. The circumflex is a branch of the posterior cord of the plexus and, with the sub-scapular nerves which supply the latissimus and teres major, may suffer equally with those muscles supplied by the musculo-spiral nerve in lesions of the posterior cord in the neck.

Musculo-spiral Nerve

This nerve is the termination of the posterior cord and is the largest nerve entering the arm. It winds round the back of the humerus in the musculo-spiral groove, closely applied to the bone, and is frequently damaged in fractures of the humerus, particularly in gunshot wounds. In such cases paralysis may be immediate from the nerve being torn by the bullet or by the sharp fragments of bone, or the signs of paralysis may develop two or three weeks later, due to the nerve being involved in scar tissue or in callus, when an operation may be necessary to free the nerve. Complete severance of the nerve will require suture of the divided ends, which is sometimes very difficult or even impossible, on account of retraction of the ends when secondary suture has been delayed. It is all-important therefore to diagnose complete rupture of the nerve early, as primary suture gives much better chance of recovery.

The nerve is sometimes pressed upon as it winds round the bone by a crutch used for leg injuries, often called crutch palsy, or it may be similarly injured by pressure during the administration of an anæsthetic, the arm hanging over the sharp edge of the operating table, or the pressure may occur during sleep, often of intoxication, as when sitting in a chair with the arm thrown over the wooden back, or from the pressure of the head lying on the outstretched arm. This latter cause, owing to the frequency of the discovery of the paralysis on Sunday morning, has often been called Saturday night or Sunday paralysis.

Chronic alcoholism is a predisposing cause to neuritis and musculo-spiral palsy, and the latter is sometimes found in alcoholics without any definite evidence of prolonged pressure. The extent of the paralysis varies somewhat according to the site of the damage, and the triceps muscle will be paralysed when pressure is exerted high in the axilla, as in crutch palsy or by the head of the bone in dislocated shoulder, but when the nerve is compressed by the edge of the table while the patient is under an

anæsthetic or during sleep, the triceps escapes almost entirely, and the paralysis is then limited to the supinator longus and to the extensor muscles of the fingers, thumb and wrist, the resulting wrist-drop being easily recognised.

Numbness and partial anæsthesia will also be found over the distribution of the radial nerve on the outer side of the back of the hand and below the wrist, extending down to the thumb nail and to half-way along the first phalanx of the index and middle fingers. Within a few days electrical changes appear, and the reaction of degeneration will become evident in proportion to the degree of the nerve damage. In the most severe cases of rupture of the nerve both faradic and galvanic irritability of the muscles disappear completely, no contractions to faradism being obtainable after a week, and after two or three weeks only to reversals of very strong currents, such as 25–30 milliamperes, will galvanic reactions be visible.

In these complete cases such as crutch or sleep palsy, hyperexcitability to galvanism will become evident during the second week, with complete polar changes of reaction of degeneration; in still slighter cases where the nerve is partially involved in scar tissue, though there may be considerable wrist- and finger-drop, yet the faradic reactions may be retained but diminished and somewhat sluggish.

The musculo-spiral nerve may be affected by a perineuritis of rheumatic origin, or it may be secondary to muscular over-strain, as in lifting a heavy person on to an operating table. In these cases the principal symptom will be pain, without weakness or anæsthesia, whereas in cases of direct injury to the nerve there may be no pain whatever.

The pain in musculo-spiral perineuritis is usually increased by such movements as stooping with the arms outstretched, or putting on the boots. The pain is of an aching, wearing type and may last for many weeks. Rest of the arm in a sling is advisable, and radiant heat applications and anodyne liniments may be ordered, but massage should be avoided. For the wrist-drop in musculo-spiral

paralysis means should be adopted for keeping the wrist and fingers more or less passively extended.

This should not be done by fixing the wrist and fingers in complete extension by means of bent splints and bandages. During the War I saw numbers of such cases in which irreparable damage had been done by this treatment being continued for weeks, setting up arthritis and permanent fixation of the wrist joint.

The best means is the use of an elastic glove extension such as is made for me by Henry Lewis, 2, Westmoreland Street. In principle it consists of a leather glove with the fingers cut off at the first interphalangeal joint; tapes are sewn to the backs of the inside of the fingers of the glove at this point and, passing upwards, are collected together at the level of the wrist and firmly attached to a leather strap. This is then continued upwards along the back of the forearm as a broad and strong elastic band of four inches in length and again by a leather strap which is fixed to an armlet above the elbow, the tension being alterable by a buckle. This arrangement imitates as closely as may be the action of the extensor muscles and tendons, and permits the use of the hand while yet maintaining some extension of the wrist and fingers.

One object of this relaxation apparatus is to keep the paralysed muscles from being constantly over-stretched, which would happen if the wrist-drop were permitted to remain untreated, for, as Sir Robert Jones showed, paralysed muscles take very much longer to recover when persistently over-extended.

Musculo-cutaneous Nerve

This nerve is a branch from the anterior portion of the brachial plexus, and is one of the divisions of the so-called outer cord, its nerve fibres being derived from the fifth and sixth cervical nerves. It supplies motor innervation to the main flexors of the forearm, the biceps and brachialis anticus muscles, and sensation to a strip of skin along the radial side of the forearm as far as the wrist. Paralysis

of this nerve will therefore seriously weaken the power of the elbow joint, the only remaining flexor action being due to the supinator longus, and to a part of the brachialis anticus which receives fibres from the musculo-spiral nerve. This nerve is not commonly injured, but may suffer from pressure by the head of the humerus in dislocated shoulder, and perforating wounds by gunshot or stab occasionally involve it.

FIG. 15.—Anæsthesia in case of gunshot injury of left musculo-cutaneous nerve.
b. Loss to light touch and prick.

MEDIAN NERVE

The median nerve, like the musculo-cutaneous, is derived from the anterior portion of the brachial plexus, being made up of two branches or heads, one from the outer cord and one from the inner cord, which meet and join over the axillary artery. It derives its nerve fibres from four spinal segments from the sixth cervical to the first dorsal. The outer head carries fibres from the fifth and sixth cervical nerves to supply the pronator radii teres and the flexor carpi radialis muscles, and also the cutaneous dis-

tribution of the median to the thumb and outer three fingers.

The inner head derived from the inner cord carries no cutaneous nerve supply, but motor fibres to the flexors of the fingers and the muscles of the thenar eminence and outer two lumbricals. In rare cases the median nerve also innervates the first dorsal interosseous muscle and a partial supply to the second dorsal interosseous. The median nerve gives off no branch above the elbow, and it lies close beside the brachial artery and its concomitant veins. It enters the forearm between the two heads of the pronator radii teres, and then gives off muscular branches to all the flexors of the fingers and wrist except the flexor carpi ulnaris and the inner half of the flexor profundus digitorum, which are supplied by the ulnar nerve. The nerve crosses in front of the brachial artery near the elbow, and in the upper part of the forearm lies deeply, protected by the flexor muscles, but as it nears the wrist it lies more superficially, between the tendons of the flexor carpi radialis and the palmaris longus.

Sensation.—The median cutaneous supply is over the distal portion of the palm of the hand as far as the ring finger, and also the palmar surface of the thumb, the first and second fingers, and the outer half of the ring finger. It also supplies sensation to the dorsum of the first and second fingers and the outer half of the ring finger as high as the first interphalangeal joint, or even slightly higher. Occasionally in median nerve lesions anæsthesia may also be found on the dorsum of the last phalanx of the thumb.

Sensation should be tested for light touch, as with a camel's hair brush, for pain sensibility with a steel pin, and for pressure by a blunt pencil, or even a hard squeeze. Temperature sense should be tested by the application of test-tubes containing water at 100° F. and at 55° F. Other tests of sensation employed in certain cases are vibration of a tuning fork, the spacing of compass points, the recognition of weights, shapes of objects, etc.

The nerve fibres supplying the skin on the dorsum of the

digits probably run in the dorsum of the nerve. Total median nerve paralysis causes not only loss of tactile sensations over this area, but also the perception of pressure is lost, the hardest squeeze on the ends of the first and second fingers being quite imperceptible. Paralysis of the median nerve causes some weakness of pronation of the wrist together with complete inability to flex the thumb or index finger, though the other three fingers can be bent; the wrist too in flexion will fall to the ulnar side. There will be flattening also of the thenar eminence owing to wasting of the abductor and opponens pollicis, and the outer head of the flexor brevis, so that proper opposition and abduction of the thumb are lost.

The median nerve may be injured by a dislocated shoulder, and I have seen this injury occasion most intense causalgia in the palm of the hand.

The nerve may also be damaged in the arm by perforating wounds or by the pressure of a tourniquet. It is rarely injured in the upper part of the forearm, but as it nears the wrist it may be cut across by a slash from a knife or by broken glass, when the flexor tendons suffer simultaneously.

I have also known it punctured by a hypodermic needle and injected accidentally by fibrolysin. A tight bandage and splinting of the forearm may similarly injure the median nerve slightly, so that symptoms of median neuritis may accompany *ischæmic myositis* or Volkmann's contracture, which is due to a primary muscular rigor mortis produced by the tight bandage preventing blood and lymph circulation in the muscles. This latter condition is recognised by primary flexor contracture developing within perhaps 24 hours of the bandaging, thus differing from paralytic contracture, which takes much longer to develop. Prolonged intermittent pressure on the palm of the hand by leaning heavily on a stick may damage the sensory nerve terminations of the median nerve in the palm, giving rise to numbness, tingling and anæsthesia of the thumb and outer fingers.

Intermittent pressure on the ball of the thumb by an instrument such as a trowel-handle in gardening, or by a

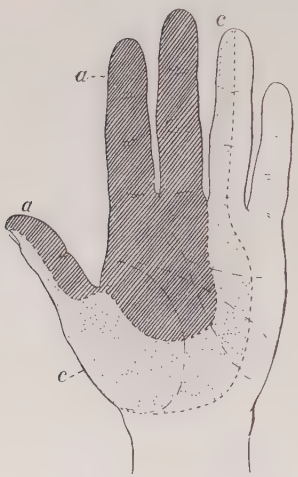

Fig. 16.—Palm.

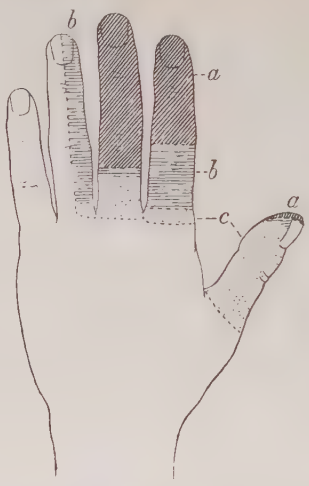

Fig. 16*a*.—Back.

Anæsthesia in a case of divided median nerve (palm).

a. Total anæsthesia to touch, prick and pressure.
b. Loss to light touch and prick.
c. Diminution to light touch and prick.

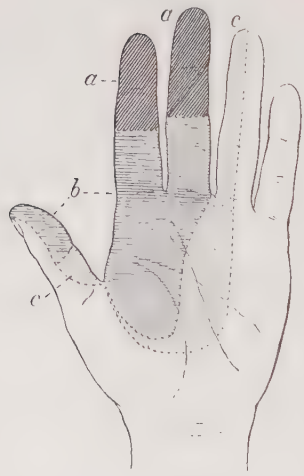

Fig. 17.—Palm.

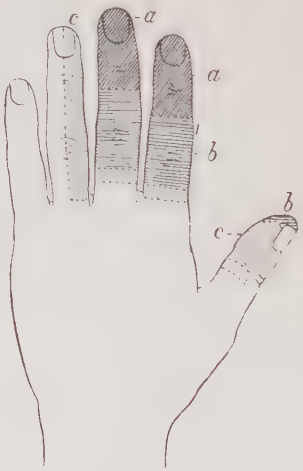

Fig. 17*a*.—Back.

The same patient as in Fig. 16, seven weeks after suture of the nerve. Note the shrinkage of the area of total anæsthesia.

a, *b*, *c*, as in Fig. 16.

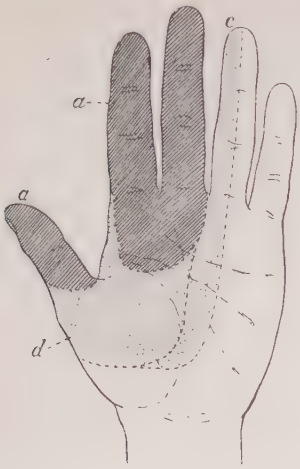

FIG. 18.—Palm.

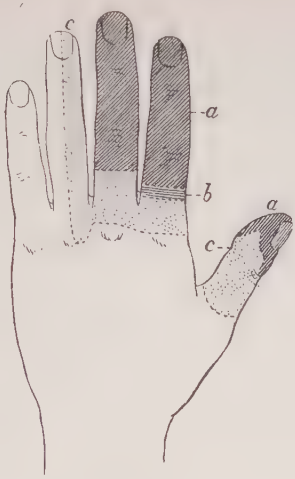

FIG. 18*a*.—Back.

Another case of divided median nerve, to illustrate the variable area of anæsthesia, cf. Fig. 16, and also anæsthesia on dorsum of thumb.

a, *b*, *c*, as in Fig. 16. *d*. Considerable diminution to all forms of sensation, but prick just felt.

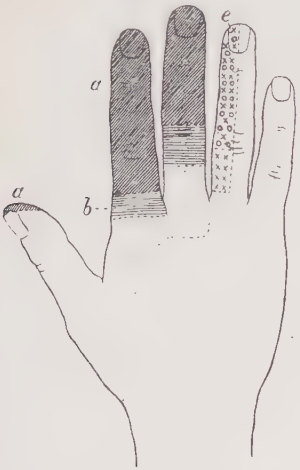

FIG. 19*a*.—Back.

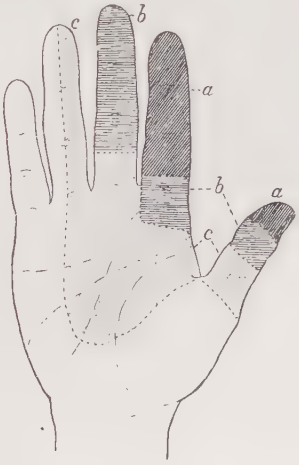

FIG. 19.—Palm.

Partial division of right median nerve, about three-quarters of the nerve being divided, leaving a bridge of uncut nerve tissue anteriorly. Note the area of partial anæsthesia on the palmar surface of the middle finger, corresponding to the uncut portion of the median nerve.

a, *b* as in Fig. 16, *c*, *d* as in Fig. 18. *e*. Slight analgesia, with sensation of soreness.

scrubbing brush, may cause wasting of the thenar muscles supplied by the median, through pressure neuritis of the nerve branches. This is a form of occupation neuritis.

Extreme wasting of the opponens and abductor pollicis may be seen sometimes in arthritis of the trapezio-metacarpal joint at the root of the thumb, and reaction of degeneration will be found, indicative of a motor neuritis. Pain is always a feature of these cases, referred to the outer side of the wrist, and even to the elbow, with tenderness to pressure on the dorsal side of the joint. There will be no anæsthesia, and no wasting of the first dorsal interosseous muscle, which latter point is almost certain to be present in muscular atrophy of the hand of spinal origin.

The median nerve is only rarely damaged by pressure during sleep or under a general anæsthetic. The nerves that usually suffer such pressure paralysis are the musculospiral and occasionally the ulnar and the external popliteal.

Causalgia

Injuries of the median nerve in any part of its course between the axilla and the thenar eminence are liable on rare occasions to be followed by a peculiar burning pain in the hand, more or less continuous, but with paroxysms often of intense severity at varying intervals. Causalgia[1] is a term suggested by Weir Mitchell in 1864 to describe such cases of intense pain in various injuries of nerves which he met with during the American Civil War. Denmark in 1813 described the same condition in a soldier wounded at Badajoz. Very rare in civil practice, this condition was almost forgotten until the recent Great War, when a large number of cases were observed resulting from gunshot injuries. This curious condition of causalgia is almost limited to injuries of the median and internal popliteal nerves, the burning pain and other symptoms presently to be described affecting the palm of the hand and the sole of the foot.

[1] Gr. καῦσις, a burning.

Indeed it has been suggested that it is the preponderating cutaneous supply for sense organs for touch and pain in the palm and sole that determines the practical limitation of causalgia to these regions.

Though during the War I saw many scores of cases of causalgia due to median and popliteal nerve lesions, only very infrequently did I meet with it affecting other nerves, and then with less severity than is common with the former

FIG. 20.—Causalgia due to gunshot wound which divided the internal cutaneous nerve and produced light scar tissue compressing the ulnar nerve. Notice the wide area of hypersensitiveness on the hand, indicated by large spots.

nerves. In many cases the evidences of nerve injury are very slight, scarcely any anæsthesia or wasting being detectable, though occasionally dense anæsthesia may be present. Usually the hand is held semi-flexed and protected by a sling and thick cotton wool, owing to intense hyperæsthesia and tenderness to the slightest touch. The skin, often perspiring profusely, is mottled, red in colour, the finger pads are slightly wasted and at times the patient keeps the hand continually wet with cold water to relieve the pain, which is of a burning character more or less constant. The pain is liable to be exaggerated by any sudden jolt to

the limb or by any sudden emotional shock, often of trivial character, either auditory or visual, such as a banged door or even merely seeing a bird dart out of a hedge. The severity of the pain may prevent sleep, and may reduce the patient to an intensely neurasthenic state, so that relief of the pain becomes an urgent necessity.

During the War injuries producing causalgia were practically always gunshot wounds, but the actual damage to the nerve producing the symptoms is not fully understood.

It has been suggested that the explosive effect of a high-velocity bullet which is not in close proximity to the nerve trunk, even without touching it may produce acute vascular effects such as thrombosis and minute hæmorrhages in the nerve sheath or in the nerve trunk itself.

Exploratory operations for the relief of this condition may disclose no naked-eye damage to the nerve trunk, but more frequently some laceration of the nerve is found, and scar tissue adherent to the nerve with some wasting of the nerve trunk below. In civil practice I have met with only two cases of marked causalgia, one due to laceration of the thenar eminence by machinery, and the other from a dislocated shoulder in an old lady of 74, due to pressure of the head of the humerus in the axilla, the pain following instantly upon the dislocation.

Treatment.—This has been mainly empirical owing to the doubtful pathology of the condition. In the slighter cases light massage may be bearable, and if persisted in a cure may result; in many, however, the tenderness and hyperæsthesia are much too great to permit of any handling.

Electricity is worse than useless. Many patients get relief from cold applications or even from continual dropping of cold water on the hand.

In some hot fomentations ease the pain, and anodyne liniments such as opium and belladonna should be tried. In the more resistant cases operative treatment must be undertaken. Exploration of the wound may disclose scar tissue nipping and encircling the nerve, in which case the

scar tissue should be dissected off and the nerve enclosed in fat as a protection against the scar reforming. If no improvement results in a fortnight the wound must be reopened and the nerve again defined to a point well above the injury. Injection of the nerve at this level with ninety per cent. alcohol, if done thoroughly, will block all nerve impulses from passing up or down so that complete anæsthesia and paralysis will ensue.

Many cases have received immediate relief by this means, but in some cases the pain continues quite unaltered, although the nerve has now no power of conduction. Indeed I have seen several causalgias of the median nerve quite unaffected by complete nerve blocking by alcohol injection of the nerve above the injury, with subsequent resection of the whole brachial plexus within three months of the original injury. Possible explanations of such continuance of the pain are (1) hysteria, (2) a central functional effect upon the pain centres in the optic thalamus set up by the injury, resulting in persistent pain. Probably a psychic element enters into all such cases, though the suffering indeed seems most distressing.

Such pain may be compared to the acute mental distress of some anxiety neuroses and melancholias, where there is no question of any organic injury. One view of the pathology of causalgia which has been advocated is that it is due to a vasomotor neuritis, and operations suggested by Leriche have been done to strip off the sympathetic nerve plexus around the brachial artery, with apparently varying success. It should not be forgotten, however, in appraising such results, that practically any treatment recommended for any disease will have its enthusiastic supporters.

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Ulnar Nerve

The ulnar nerve, like the median, gives no branch above the elbow. It is formed from the first dorsal and eighth cervical nerves, which unite to form the inner cord, and this receives a branch, sometimes of moderate size, from the seventh cervical nerve which perforates the inner head of the median to reach the ulnar, a branch I have named the outer head of the ulnar (p. 74).

When the inner cord suffers as a whole, which it may do in Klumpke paralysis, there are weakness and wasting of all the muscles in the hand and loss of flexion of all the fingers and last joint of the thumb, with anæsthesia of the little finger and inner half of the ring finger and of the inner portion of the hand, with a strip of anæsthesia of the inner side of the forearm up to just above the elbow. The strip from the wrist upwards is the area supplied by the internal and lesser internal cutaneous nerves, the ulnar itself not supplying the section above the styloid process of the ulna. With the exception of pressure paralysis in the axilla by a dislocated humerus, the ulnar nerve is rarely damaged above the elbow unless by direct injury from a bullet or stab wound.

Fracture of the humerus, though it frequently damages the musculo-spiral nerve, rarely affects either of the other nerve trunks in the arm, except in very severe injuries, as in crushes by machinery. Occasionally, however, prolonged application of a tourniquet round the arm has been known to damage all the nerve trunks, causing weakness of the wrist and finger movements and anæsthesia of the hand. The ulnar nerve may suffer near the elbow joint in various ways, first by direct violence from fracture of the olecranon and fractures into the elbow joint; secondly, an ulnar paralysis may be set up through prolonged flexion of the elbow, as when the arm is strapped to the chest for fracture of the clavicle.

The neuritis set up by such pressure is usually not severe, merely causing tingling and numbness and soreness of the

inner portion of the hand. Prolonged flexion of the elbow together with pressure, such as are caused by sleeping with the head leaning on the hand, with the elbow resting on a table, may cause complete ulnar paralysis. This is one of the forms of " sleep palsy " or pressure neuritis. The resultant paralysis causes weakness of flexion of the wrist to the ulnar side, flexor palsy of the little finger and paralysis of all the interossei and the two inner lumbricals. In rare

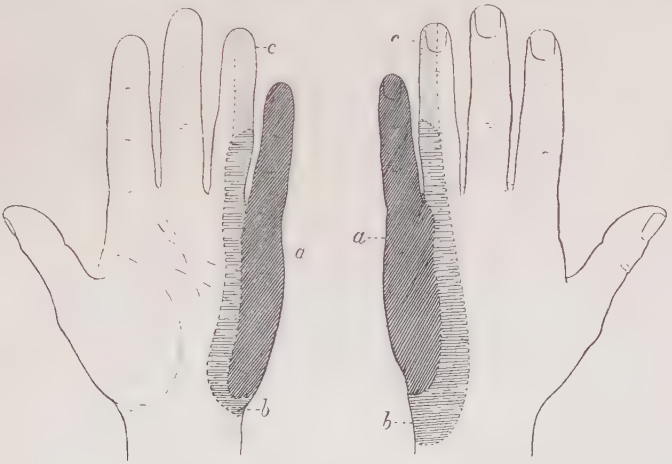

Fig. 21.—Palm. Fig. 21*a*.—Back.

Divided left ulnar nerve.

a. Total anæsthesia to touch, prick and pressure.
b. Loss to light touch and prick.
c. Diminution to light touch and prick.

cases the first dorsal interosseous, and even the second, may receive its motor supply from the median nerve, and will not then be affected in ulnar paralysis. Reaction of degeneration, partial or complete, will be found in proportion to the severity of the injury to the nerve.

Sensation.—In cases of aseptic clean division of a nerve, there will be no trophic wasting of the skin or nails, no wasting of the finger-pads nor glossy skin, nor signs of vasomotor disorder or sweating of the anæsthetic area. Such signs are evidence of an irritant neuritis, such as

occurs with incomplete destruction of the nerve, with sepsis of the wound. The skin will, however, be dry, and usually becomes thickened and almost scaly from epithelial detritus.

In total ulnar paralysis there will be complete anæsthesia to all forms of sensation, including pressure over the whole of the little finger, and generally of the inner border of the hand for an inch or two above the little finger. There will be loss to light touch and to pin-prick and to medium

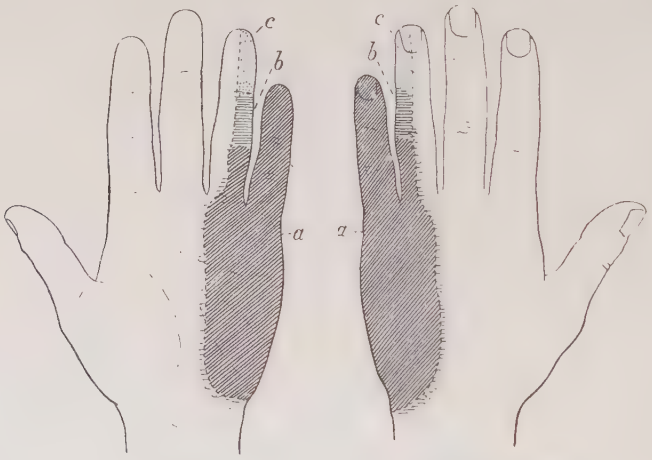

FIG. 22.—Palm. FIG. 22*a*.—Back.

Divided left ulnar nerve illustrating variation of larger area of total anæsthesia.
a, *b*, *c* as in Fig. 21.

temperature changes over the inner half of the ring finger, and over the inner border of the hand as high as the wrist, and extending outwards usually as far as the cleft between the middle and ring fingers. In rare cases the variation of sensory cutaneous supply on the hand of the median and ulnar nerves may amount to a whole finger. I have seen gunshot wound of the median produce anæsthesia of the thumb, index and half the middle fingers only, and, as a complement to that, in another case of division of the ulnar nerve the anæsthesia extended outwards as far as the centre of the middle finger, the whole of the ring

finger, as well as the little finger and inner border of the hand being totally insensitive to prick or to hard pressure.

Figs. 21, 22, 23 illustrate variations in the extent of anæsthesia in complete ulnar nerve lesions.

Though there is no anæsthesia to be detected on the outer side of this area, yet, in cases of irritant neuritis of the ulnar such as causalgia, there will be found a zone of hyperæsthesia and pain extending outwards as far as the

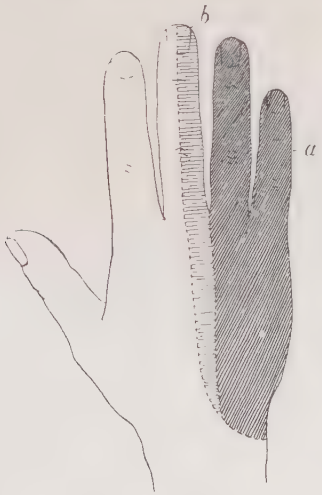

FIG. 23.—Back.

Divided right ulnar nerve. Note the abnormally large area of total anæsthesia, including the whole of the ring finger, and nearly complete even to pressure on the inner half of the middle finger.

a, *b* as in Fig. 21.

ball of the thumb and the root of the index finger, with perhaps evidences of vasomotor disturbance, such as redness and mottling of the skin, and sometimes excessive sweating (see Fig. 20). In such a case the little finger will show most intense signs of trophic disturbance, such as glossy skin, wasting of the finger-pad and incurving of the nail.

Chronic Ulnar Neuritis is found commonly in anæsthetic leprosy, the nerve being notably thickened at the elbow joint. Similar thickening may also be met with

in the rare conditions of hypertrophic interstitial neuritis described by Dejerine and Thomas, and in hypertrophic polyneuritis. Ulnar neuritis is also met with in chronic diabetes.

Dislocation of the ulnar nerve at the elbow has occurred in cases where the condylar groove is unduly flat, with resultant pain, tingling and partial paralysis.

Chronic neuritis of the ulnar nerve may be set up just above the elbow joint owing to a chronic periostitis in the ulnar groove; this may arise through constant pressure or by repeated blows upon the elbow, or it may be a late result many years after an injury to or inflammation around the elbow joint. The symptoms produced will be gradually increasing tingling of the little finger, and numbness and pain referred to the inner part of the hand even on slight flexion of the elbow, and the nerve may be found considerably thickened and enlarged. Cramp too of the hand when used for such movements as carrying or writing may be an early sign in a sub-acute neuritis of the ulnar, just as may occur in cases of rib pressure upon the first dorsal nerve. It is common for many persons to complain of such symptoms if the elbows are kept flexed for more than a few minutes, as the nerve is stretched round the elbow joint by such flexion. This common disability is a result of the adoption of the erect posture by human beings and the consequent altered use of the fore-limb from a locomotive to a prehensile organ, the nerve not yet having adapted its position to the changed use and greater flexion of the elbow joint. The treatment of this form of chronic neuritis of the ulnar is to liberate the nerve from the back of the elbow and bring it round to the front of the joint by dividing the inner head of the flexor carpi ulnaris. Such a position for the nerve is far better adapted for modern conditions of life, though it is unlikely ever to be developed naturally on Darwinian principles of natural selection.

Occupation Neuritis of the ulnar is not common, but has been met with after practising the piano for many hours daily, and in wood carvers and other trades. The

symptoms are usually slight –tingling and numbness of the two inner fingers, with perhaps cramp on flexion of the fingers, and some weakness of the interossei.

With the exception of occasional partial damage from tight bandaging, when the resultant ischæmic myositis or Volkmann's contracture may be accompanied by signs of neuritis of both median and ulnar nerves, the ulnar nerve is rarely injured in the forearm until it nears the wrist, where it becomes comparatively superficial. A frequent injury is from broken glass, by the hand being dashed through a window, various tendons and the ulnar nerve being cut. The median nerve too may suffer from such an injury. If the ulnar nerve is divided below the point where the dorsal cutaneous branch is given off at the junction of the middle and lower thirds of the forearm, then there will be no anæsthesia on the back of the inner side of the hand and fingers, but only of the palmar surface, together with paralysis of the interossei, two inner lumbricals, the hypothenar muscles, the adductor pollicis and the inner half of the flexor brevis pollicis. Owing to this paralysis wasting will soon become evident between the metacarpal bones, and the hand will tend to become "clawed," the first phalanges being hyper-extended and the two terminal phalanges flexed owing to the unopposed tonic action of the extensor communis and of the remaining flexors of the fingers supplied by the median nerve.

Complete ulnar paralysis is very disabling to the use of the hand, on account of the anæsthesia of the little finger rather than on account of any muscular paralysis, and any treatment which may restore sensation to the little finger and inner side of the hand will be of immense value, even though the wasted interossei do not recover at all, which is indeed a common sequel. Primary suture of the ulnar nerve, when it is divided along with cut tendons in wounds above the wrist, will give the best chance of the most complete recovery, if the wound heals cleanly without any suppuration. A septic wound, however, is apt to set up troublesome neuritis and the development of contractile

scar tissue, which may prevent any recovery. A secondary operation may be considered later on when all inflammation and sepsis have disappeared, and the problem will be presented of how to unite healthy nerve above to a wasted nerve below after cutting out the diseased portion. Nerve grafts have been tried, but have usually failed, though I have seen success rapidly follow grafting one and a half inches of a cat's sciatic nerve between the two ends of the ulnar above the wrist. In this case sensation returned in the little finger, though the interossei and hypothenar muscles remained wasted. Even with the most carefully done primary suture recovery is never perfect in a cut mixed motor and sensory nerve such as the ulnar, some tactile anæsthesia being always present and the finer discrimination known as epicritic sensibility being diminished in the little finger. The same statement applies to the median, musculo-spiral, sciatic or any other mixed nerve which has been severed by an injury, as it is naturally impossible accurately to unite all motor and sensory bundles, cross union of many being inevitable, with consequent persistent partial anæsthesia and motor weakness.

The **Dorsal Cutaneous Branch** of the ulnar may suffer from prolonged pressure due to the constant wearing of a wrist watch. Stopford[1] has recorded two such cases of pressure neuritis of this branch, causing numbness and tingling, with diminished sensibility of the little finger and the inner side of the hand.

The **Deep Palmar Branch** of the ulnar may be wounded by a fall upon the palm of the hand, the nerve being punctured by a piece of glass or other sharp object piercing the hypo-thenar eminence. The result will be motor paralysis of the interossei, with clawing of the hand, but no anæsthesia, and escape of the hypothenar muscles.

Treatment of ulnar neuritis and injuries of the ulnar nerve will be on the same general lines as for other mixed motor and sensory nerves. Hot saline arm baths, ionisation, labile galvanism, and massage in the chronic stages

[1] *Lancet*, 1922, i, 993.

may be of great service in reducing pain and tendency to cramp.

Operation may be of value in cases of chronic neuritis associated with periosteal thickening and pressure on the nerve in the condylar groove at the elbow, and in cases of wounds involving the nerve, or callus from fractured bone. Aneurysm or cysts may also press upon and damage the nerve. Secondary operations, when the nerve has incompletely recovered after a stab or gunshot wound, require careful diagnosis and consideration.

If all sepsis has disappeared and recovery of function is poor, operation may disclose a partially severed nerve half strangled in dense scar tissue. Operation even two or three years after the injury may then lead to much recovery of function by dissecting away the scar and freshening the divided ends of the nerve and re-suturing.

In other cases chronic pain in the hand may be associated with tender bulbs on the nerve at the site of injury. These may be cut out, but often the relief is only temporary, lasting a few months. Even division of the nerve high up and burying its end in fat or muscle may fail to arrest the pain.

Differential Diagnosis.—Ulnar paresis has to be distinguished from rib pressure on the first dorsal nerve and from spinal diseases such as poliomyelitis and syringomyelia.

The clawed hand resulting from interosseal wasting in ulnar nerve paralysis may be mistaken for the clawed hand of progressive muscular atrophy, or syringomyelia. In the former of these two diseases the stress of the wasting is usually on the thenar eminence, so that the ball of the thumb is wasted, and the first metacarpal bone falls into line with the metacarpals of the fingers. The clawing of the fingers in progressive muscular atrophy may be more pronounced than that met with in ulnar palsy, owing to the escape of the two outer lumbricals in the latter condition.

In syringomyelia the wasting may closely simulate that of ulnar paralysis, as in many cases of spinal gliosis the thenar eminence is affected much later than the remaining

muscles in the hand. Another point of distinction in syringomyelia is wasting of the flexors in the forearm while the extensors of the fingers and wrist remain unaffected.

In progressive muscular atrophy the wasting often skips a segment, the extensors in the forearm being involved after the hand muscles. Moreover, the sensory changes will help to determine the diagnosis, in ulnar palsy the anæsthesia involving the little finger and inner side of the hand, as already described, while in syringomyelia the sensory loss is usually dissociated, the tactile sense being scarcely at all affected, while loss of discrimination of temperature and loss to pin prick may be present over the whole hand and forearm. In progressive muscular atrophy and in acute poliomyelitis there are no sensory changes.

The **Internal Cutaneous nerve**, a branch of the inner cord of the plexus, and deriving its fibres from the first dorsal nerve, has no motor fibres and supplies a strip of skin along the inner side of the forearm from just above the elbow down to the wrist. Paralysis of this nerve alone does not cause any great disability, except from hyperæsthesia in incomplete or irritant lesions of the nerve. In cases of rib pressure or other lesions of the first dorsal nerve there will be diminished sensibility on the inner side of the forearm in addition to the complaint of pain, and atrophy of the intrinsic musculature of the hand. Lesions of the inner cord of the plexus will also involve the internal cutaneous nerve fibres.

Functional Muscular Atony in its extreme form is a remarkable condition of flaccid paralysis of muscles in one upper or lower extremity, resulting usually from shock, such as an injury to the limb, and for this reason it is liable to be confounded with nerve injuries. A certain amount of general wasting of the muscles occurs, and there is some loss of sense of position, but as a rule no cutaneous anæsthesia is present.

Vasomotor paresis is obvious, the limb being cold and slightly blue, but no trophic changes are seen, such as wasting of the finger pads, incurving of the nails, glossy

skin or joint adhesions. There is no pain, and the limb hangs useless, with little or no power of movement except perhaps slight flexion of the digits occasionally, the patient declaring that he has no idea how to move the parts. The muscles are very flaccid, but the deep reflexes are maintained, and the electrical reactions to faradism persist unchanged. A form of electrical change which I have named the *cyanotic reaction* [1] may be well marked when

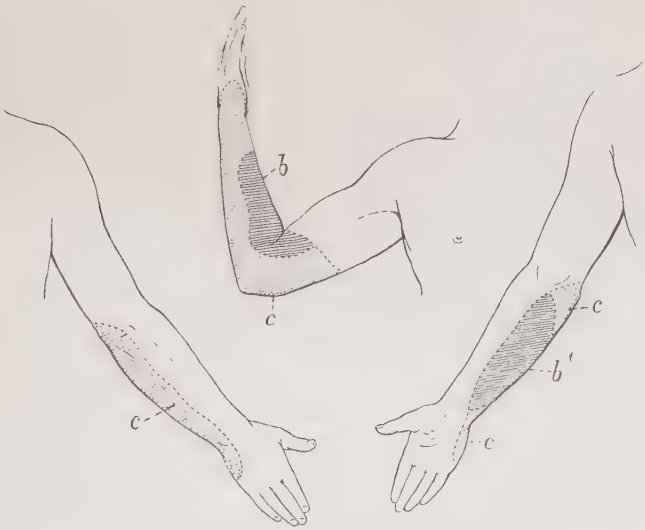

FIG. 24.—Divided right internal cutaneous nerve.
b. Loss to light touch and prick.
c. Diminution to light touch and prick.

the limb is cold and blue, as is also seen in Raynaud's disease and other causes of cyanosed extremities. The reactions both to faradism and galvanism are then very sluggish, but after soaking the limb in hot water for a few minutes the normal brisk twitch of the muscles both to faradic and galvanic shocks will reappear.

An officer, æt. 21, in Mesopotamia in Jan. 1916 was shot through the left upper arm, fracturing the humerus near the head. The arm was totally useless from the

[1] Harris: "Electrical Treatment," 1908, p. 137.

first. The fractured bone healed well, but little or no recovery took place in the paralysed limb. When I saw him in May 1916 the left arm hung flaccid and useless, the fingers were bluish, even cyanotic when the arm was kept hanging down in cold weather. The arm measured $1\frac{1}{2}$ inches less round the mid biceps than the right arm, all the muscles being flabby. There was no trace of anæsthesia, though he complained of loss of sensation of position of the hand, and of some hyperæsthesia of the finger-pads. All the muscles reacted to faradism equally, and the galvanic reactions also were normal, though all the contractions were distinctly weaker than in the other arm. At first there was not a trace of movement in the arm or fingers, but after daily faradism and encouragement slight movements of the fingers and of the biceps returned, though even after the lapse of seven years there was little change in the condition, and the arm was quite useless.

The case was sent to the hospital originally as one of musculo-spiral palsy, though only slight examination was necessary to show the weakness was wider in extent. Neuritis due to ordinary nerve injury was excluded by the presence of good faradic reactions and absence of reaction of degeneration, and the proof of a strong "functional" element in the weakness was provided by the fact of several muscles situated above the lesion being equally paralysed with the rest of the arm. Thus the deltoid, the latissimus dorsi, and even the movement of shrugging of the shoulder by the trapezius were completely paralysed for voluntary effort, though on coughing the latissimus contracted strongly. Malingering is at once excluded by the wasting and the cyanosis, but the pathology of the condition is obscure.

CHAPTER V

NEURITIS OF LOWER EXTREMITY. SCIATICA

Nerves of the Lower Extremity

Two important nerves supply the greater portion of the lower limb, the anterior crural and the sciatic, though lesser *rôles* are played by the obturator nerve, the external cutaneous and the genito-crural. The anterior crural nerve is derived from the upper part of the lumbo-sacral plexus, its fibres belonging mostly to the third and fourth lumbar segments. The ilio-psoas mostly derives its fibres from the first and second lumbar nerves, which may be damaged by psoas abscess secondary to tuberculous Pott's disease of the spine.

Growths too may involve these nerves, or acute staphylococcal infection secondary to infective periostitis of the vertebræ may damage the lumbo-sacral plexus.

Anterior Crural Nerve.—This nerve derives its fibres from the third and fourth lumbar nerves, and, leaving the pelvis after its exit from the psoas muscle, passes beneath Poupart's ligament on the outer side of the femoral artery, and soon divides into muscular and cutaneous branches. This nerve supplies all the muscles on the front of the thigh except the tensor fasciæ femoris, which is supplied by the superior gluteal nerve.

With the deep muscular branches are given off two articular branches to supply the knee-joint, which, it will be remembered, is also supplied by a branch from the obturator nerve. The cutaneous branches are three, the middle cutaneous, internal cutaneous, and the long saphenous, the two former supplying the integument on the front and inner side of the thigh down to the knee, while

the long saphenous, the longest of the three branches, supplies the inner side of the leg as far forward as the crest of the tibia and down to the inner side of the foot and ankle.

Anterior Crural Neuritis

The anterior crural nerve may be damaged within the pelvis by pressure of a tumour, or by injury from a stab or other perforating wound. Tumours such as periosteal growths of the ilium or of the lumbar vertebræ invading the psoas are liable to damage the anterior crural nerve, causing severe pains down the front of the thigh and leg, which must be distinguished from sciatica.

The knee jerk will be lost, but the Achilles jerk will be unaffected, the reverse obtaining in sciatica. The nerve may also suffer from neuritis in chronic diabetes, in which case there is usually a considerable amount of aching pain on the front and inner side of the thigh down to the knee, and the patient may complain of soreness and pins and needles over this area.

Usually in diabetic neuritis of the anterior crural nerve there is little or no motor paralysis of the quadriceps, and the symptoms gradually disappear after three or four months, though the glycosuria may continue.

Influenza sometimes is followed by anterior crural neuritis, and the pain may be excruciating for a few days, rendering sleep impossible without repeated doses of morphine. Anæsthesia rarely results from this neuritis, though the knee jerk will be lost, and some motor weakness and flabbiness of the extensors of the knee may persist for many weeks, and even fibrillation of the quadriceps muscle may be observed.

I have seen a sharp attack of anterior crural neuritis follow within a week a severe burn on the palm of the hand, no other cause than the sepsis being apparent.

In paralysis of the anterior crural nerve as the result of injury or involvement of the nerve in a growth, the motor palsy is serious on account of the loss of power in the quadriceps extensor femoris. There will be weakness or

even total loss of power of extension of the knee, so that walking will be grossly interfered with, especially upstairs or uphill.

The gait in cases of quadriceps paralysis is characteristic, the patient being forced to throw his weight perpendicularly over the hip and knee on the affected side with each step taken by that foot. This produces a waddling, or rolling gait when both quadriceps muscles are paralysed, as is seen in many cases of myopathy. Though walking in a modified manner is possible, running is quite impossible when even one quadriceps is paralysed.

Any branch of the anterior crural nerve, motor or sensory, may be injured separately, the long saphenous nerve frequently suffering alone, and I have seen causalgia of this nerve resulting from a gunshot wound of the thigh.

The **External Cutaneous Nerve** is a branch of the first and second lumbar nerves and passes under Poupart's ligament into the thigh immediately beneath the anterior superior spine of the ilium, dividing into anterior and posterior branches. The anterior branch passes through a fibrous channel formed in the fascia lata, which it finally pierces, reaching the surface about four inches below Poupart's ligament, and supplies the anterior and outer surfaces of the thigh as far as the knee. As it passes through the fibrous tunnel in the fascia lata, this anterior branch of the external cutaneous nerve may become constricted by fibrous contracture, giving rise to symptoms of burning pain, paræsthesiæ and numbness along its distribution over the middle and lower portion of the front of the thigh.

This condition has been called "meralgia paræsthetica," and the symptoms may be relieved by a small operation slitting up the channel in the fascia, thereby releasing the pressure upon the nerve. This disease is, however, often wrongly diagnosed; the last three cases that I have seen in which this diagnosis had been made turning out to be diabetic neuritis, hæmatorrhachis or intra-spinal hæmorrhage in the cauda equina and, lastly, a case of intra-

medullary spinal sarcoma. In this latter case the pain was ultimately relieved after laminectomy and division of the ascending antero-lateral tract of the opposite side of the cord.

The **Obturator Nerve** arises mainly from the third and fourth lumbar nerves, and pierces the obturator foramen, where it gives off an articular branch to the hip-joint. The nerve has an anterior and posterior branch, supplies all the adductor muscles and gives a cutaneous branch to the inner side of the leg and an articular branch to the knee-joint.

This nerve, it will be noticed, gives articular branches to both the hip and knee-joints, and for this reason hip-joint disease in its earlier stages may be overlooked because of pain being referred to the knee-joint rather than to the hip. In old chronic dorsal dislocation of the hip the head is largely kept in its false position by contracture of the adductors, and in order to relax the adductors the obturator nerve may be exposed by a small operation and the nerve put out of action by an alcoholic injection.

Sciatic Neuritis

The sciatic nerve is the largest nerve in the body, being about three-quarters of an inch in width, and it runs at the back of the hip as a thick, flat ribbon into the thigh. It is made up from the anterior divisions of the fourth and fifth lumbar and first and second sacral nerves. The nerve leaves the sacral plexus, passing out of the pelvis through the great sciatic notch below the pyriformis muscle, and then passing outward and downwards close behind the hip-joint, curving slightly to lie between the lesser trochanter of the femur on its outer side and the tuberosity of the ischium on its inner side, thence passing along the back of the thigh and being covered by the gluteus maximus first, and after by the long head of the biceps. Near the lower third of the thigh the nerve divides into its two main branches, the internal and external popliteal nerves.

Though these two nerves are usually bound up in one

sheath, and called the great sciatic nerve, the two nerves are really completely separate, and the division may take place close to the sacral plexus before leaving the great sciatic notch, and indeed they may be separated by the pyriformis muscle. The external popliteal portion is that derived from the fourth and fifth lumbar nerves, while the internal popliteal, the larger of the two nerves, gets its fibres from the first and second sacral nerves.

The external popliteal passes outwards and winds round behind and below the head of the fibula, giving two articular branches to the outer side of the knee-joint. The nerve then passes in front of the leg, where it divides into the anterior tibial and musculo-cutaneous nerves. The knee-joint, it will be noticed, thus receives nerve supply from the anterior crural nerve, from the obturator nerve and from both the internal and external popliteal nerves. The external popliteal nerve through its muscular branches supplies all the muscles in the front and outer side of the leg, its anterior tibial branch running deeply on the interosseous membrane, giving an articular branch to the ankle-joint, and finally supplying the skin of the cleft between the great and second toes. The musculo-cutaneous nerve supplies the peronei muscles and the skin of the outer and lower part of the leg and the dorsum of the foot and toes, including the inner side of the great toe.

The external popliteal, or peroneal, nerve may be said broadly to resemble the musculo-spiral nerve of the upper limb, the anterior tibial branch being comparable to the posterior interosseous nerve. The internal popliteal nerve, perhaps twice the size of the external popliteal, descends through the middle of the popliteal space, passing with the popliteal artery beneath the soleus muscle. It gives articular branches to the knee-joint and two stout nerves to the heads of the gastrocnemius muscle, which also supply the soleus, popliteus and plantaris muscles.

Below the popliteus muscle the nerve is called the posterior tibial, and passes down the back of the leg beneath the calf muscles with the popliteal vessels to reach

the ankle at the back of the inner malleolus, where it divides into the external and internal plantar nerves. At this point it lies below the artery and vein. The posterior tibial nerve supplies the tibialis posticus and the long flexors of the toes and gives a branch to the ankle-joint, and then, by means of its terminal branches, the internal and external plantar nerves, it supplies the skin of the sole of the foot and toes and the intrinsic muscles, and also articular branches to the numerous joints of the foot. The internal popliteal nerve in the lower limb may be looked upon as the homologue of the combined median and ulnar nerves of the upper limb, the external plantar being comparable to the ulnar nerve in the hand, while the internal plantar resembles the median in its distribution to the plantar surface of the great toe and the neighbouring two and a half toes.

Diseases affecting the Great Sciatic Nerve.—The lumbo-sacral plexus may suffer, close to its roots of origin from the vertebral foramina, by involvement in a malignant growth, or by gummatous neuritis, both of which are not uncommon complications, and also from rheumatic inflammation.

Sciatic neuritis due to involvement of the plexus in malignant growth is more common in the female, and is usually secondary to carcinoma of the breast or of the uterus. The sciatic pain may be uni-lateral or bi-lateral, and a curious feature in what may be called "malignant sciatica" is that pain may be severe in one limb for some weeks and then may disappear from that side while neuralgic pains then attack and become concentrated in the other leg. These pains may go on for weeks and months without actual evidence of nerve damage appearing, such as loss of Achilles jerk, muscular wasting and anæsthesia.

The appearance of an intractable neuralgia affecting one hip or leg in a woman six months or more after an apparently complete extirpation of a breast cancer is a danger signal which only too often indicates recurrence of the growth in the spine. Two or more vertebræ may become

involved, and X-ray photographs may demonstrate bony destruction in the laminæ and bodies. Angular curvature and intense tenderness on pressure will ensue when the disease attacks the dorsal region, and deposits may also be seen on the ribs, producing neuralgic pains around one side. The treatment of such neuritic pains must be entirely symptomatic. Intensive X-ray treatment and radium are of no avail here, and all that can be done is to mitigate the pain by local anodynes, radiant heat, aspirin and other analgesic drugs, and lastly morphine.

The lumbo-sacral plexus may be pressed upon as a whole by a fœtal head during the later weeks of pregnancy, causing symptoms of sciatica, and during delivery, especially when forceps are used, the nerve may be so pressed upon as to cause sciatic paralysis. The nerve too may suffer from toxic absorption in cases of chronic constipation, and especially on the right side in cases of appendicitis.

Gunshot and stab wounds may divide the nerve partially or completely after its exit from the great sciatic notch, and neuritis of the whole nerve trunk or of its branches may result from chronic diabetes, from chronic septic infection or from many of the causes that may produce a polyneuritis. Chronic inflammation of the hip-joint in rheumatoid arthritis may spread backwards to involve the sheath of the sciatic nerve as it passes over the ischium, and tuberculous caries of the sacrum or ilium may for many weeks or months be heralded only by pain along the sciatic nerve, and it is not until the fluctuant swelling of a cold abscess appears over the sacrum or in the buttock that the condition is recognised to be of more serious import than an ordinary sciatica.

Sacro-iliac disease, tuberculous or rheumatic, may simulate sciatica, or may lead to it as a complication. Prolonged pressure upon the buttock even may cause sciatic paralysis, as in coal miners lying unconscious for hours upon a hard floor after an explosion of firedamp.

Total sciatic paralysis is rarely met with except after a perforating wound from stab or bullet. The resulting dis-

ability is perhaps less than one might at first expect, the patient being still able to walk, though of course with complete foot-drop; there is paralysis of the hamstrings and total palsy of all muscles that move the ankle and toes, with complete anæsthesia, including sensibility to pressure, on the outer half of the foot.

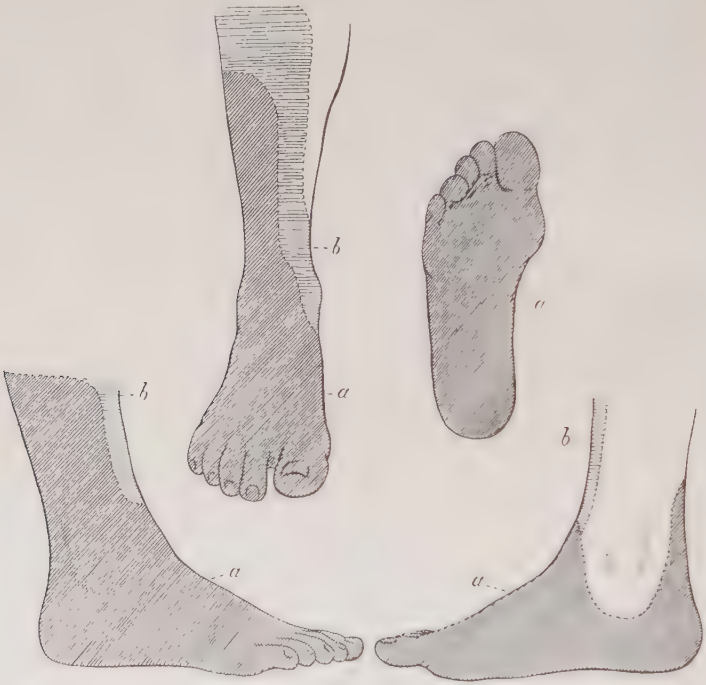

Fig. 25.—Anæsthesia in Total Sciatic Paralysis (divided right sciatic nerve).
a. Total anæsthesia to touch, prick and pressure.
b. Loss to light touch and prick.

On the plantar surface the anæsthesia is sometimes total as far as the inner border of the foot, where sensation commences to be felt through the long saphenous nerve, but occasionally sensation through this source is perceived as far as the middle of the sole. Deep sensibility, though lost over most of the dorsum of the foot, will be retained above the level of the ankle, though there will be loss to touch, pin-prick and temperature over the lower two-

thirds of the outer side of the leg as far forward as the crest of the tibia, but excluding the back of the calf and the inner side of the leg and ankle.

All the paralysed muscles will show reaction of degeneration, the foot will be cold, slightly blue and swollen, though usually not œdematous. Both the plantar reflex and the Achilles jerk will be lost, though the knee jerk will remain unaltered.

If the foot-drop is corrected by the use of a spring attachment to the boot to produce dorsiflexion of the ankle, the patient may learn to walk quite fairly, as soon as he becomes accustomed to the complete numbness of the foot. Running, however, is quite impossible, from the inability to raise the weight upon the toes, owing to the paralysis of the gastrocnemius. Clean wounds of the sciatic nerve, as from gunshot, may be treated successfully by suture of the cut ends of the nerve. No sign of recovery may show for two years or even more, and yet fair recovery of power and sensation may eventually develop, and if such an operation for suture has been done by a responsible surgeon, it is advisable to be patient in awaiting recovery, and to deny perhaps a natural curiosity to see how things are getting on by cutting down again and perhaps "freshening" the nerve ends.

Sciatica, Sciatic Neuritis.—Sciatica is a very common complaint, and in consequence there is perhaps a not unnatural tendency for the majority of people, including medical men, to affix this label to all pains around the hip and thigh whose origin is otherwise obscure. Probably the commonest error lies in overlooking arthritis of the hip, and next in importance and frequency is to mistake an anterior crural neuritis for sciatica. Psoas or sacral abscess, due to tuberculous caries, and malignant growths in the pelvis or lower lumbar vertebræ are constantly thus liable to be unrecognised in their earlier stages.

I was once asked by a physician to see as a case of sciatica an old woman in constant pain which she referred to the front of her thigh. She was lying in bed with her

knees drawn up, the knee jerk lost on the side of the pain, and a large mass easily to be felt above the groin, involving the anterior crural nerve. Such a gross mistake is, of course, due simply to want of examination, not want of knowledge, and indeed Jenner's aphorism that "more mistakes are made by not looking than by not knowing" is daily confirmed in its truth.

Such is, and probably always will be, human nature. More easily to be excused was the error of a country doctor who sent to me a lady for injection as sciatica in my consulting-room. Her pain was of a constant burning character, quite unlike any sciatica in my experience, and on examination of the thigh I found the whole of the upper portion of the femur much thickened, the growth proving to be a periosteal sarcoma.

Probably the commonest mode of onset for sciatica is an attack of lumbago, usually coming on acutely with pain across the small of the back, aggravated by stooping or sitting forwards, or by any sudden movement such as sneezing or coughing. The pain in lumbago is mostly quiescent when the patient is lying relaxed, though any sudden turning movement or sitting forward will cause the most intense pain in the back. When sciatica ensues sharp pain will be felt down the back of the hip and thigh, and within two or three days pain spreads down to the knee and below, running down to the ankle and heel and occasionally even into the foot and toes. Pain in genuine sciatica never comes round to the front of the thigh and groin, and when such pain is complained of it is usually due to some form of arthritis of the hip-joint or to anterior crural nerve disease. Though the lumbago may appear to be symmetrical in the lumbar muscles, yet it is quite rare for sciatica following such lumbago to be bi-lateral. So uncommon is it for bi-lateral neuralgic pains in the lower extremities to be due to genuine sciatica that one should always look carefully first for any signs of polyneuritis, bone disease, pelvic tumour, or central spinal disease such as tabes or spinal growth.

It is true that genuine sciatica may be bi-lateral, and I have met with many such cases, and occasionally the greater incidences of pain may alternate from one limb to the other over a number of years, the patient hardly ever being completely free of some pain. Such bi-lateral cases exhibit the same symptoms and are pathologically identical with the more common uni-lateral form.

The pain in sciatica is more or less constant, and may be extremely severe, yet varying at different times of the day. Like lumbago, the discomfort is increased by stooping, and by sudden movements, especially by sneezing and coughing. Unlike lumbago, complete rest and relaxation do not necessarily abolish the aching. Walking and standing usually aggravate the pain in sciatica, though some suffer more on sitting, especially on a hard seat; very occasionally the pain is worse on lying in bed, though the patient may be able to walk and even dance in comfort. Stretching the nerve by raising the heel from the bed, while keeping the knee straight, is usually intensely painful, and tenderness is often present at various points along the course of the nerve, more especially over the sciatic notch, in the neighbourhood of the tuber ischii, and just above the popliteal space. Sometimes the pain is so severe that the patient can get scarcely any rest or sleep, necessitating the use of opiates and other analgesic drugs.

The duration of the attacks is very variable, though acute pain preventing sleep, due to the presence of active neuritis of the nerve sheath, rarely lasts more than four or five weeks. I have seen the most intense sciatica commence shortly after exposure to chill, the pain in the leg and foot, lasting eight days, becoming so severe that sleep was impossible in spite of morphine, and then all pain disappeared gradually one afternoon and did not recur. There was no loss of Achilles jerk, or any other sign of nerve injury, the inflammation probably being limited to the nerve sheath. Such rapid disappearance of the pain in both sciatica and in acute brachial neuritis is not uncommon.

An unusual termination of an attack of acute sciatica with partial temporary paralysis of the leg I saw in an officer *æt.* 36, who developed lumbago during the early summer, the pain and stiffness increasing until September, when left sciatica appeared.

This did not become acute until after an attack of appendicitis at Christmas, the pain getting gradually more severe, until about five weeks later it was especially intense one morning when he noticed numbness coming on during half an hour while attempting to dress. Immediately all pain disappeared, except for some dull ache at the back of the thigh, but the foot remained weak. When examined three weeks later there was definite weakness of the gastrocnemius, the patient being unable to raise the heel from the ground, and there was partial reaction of degeneration in all the left leg muscles, with loss of Achilles jerk. X-ray examination showed no bony or joint disease and he afterwards steadily improved.

A very common cause of acute sciatica is chill and exposure to damp and cold, especially sitting or standing about in wet clothes; sometimes the severity of the pain may be so intense that no rest or sleep can be obtained without opiates. Acute sciatic neuritis such as this must be treated by rest in bed. The patient lies slightly turned on the opposite side, being unable to bear any continued pressure upon the buttock of the affected side. The knee is kept mostly flexed, and some relief may be obtained by a pillow placed between the knees. The softer the mattress the better, and an air mattress or even an old-fashioned feather bed is a valuable aid to comfort. On the principle that an inflamed nerve should be rested, treatment by fixing the limb and side by bandaging a Liston long splint from the axilla to the ankle has been recommended.

Such immobilisation, however, is always a failure in practice, as change of position at intervals is an absolute necessity, even though the change at the moment aggravates the pain, and any fixation by splint treatment is nothing less than refined torture. Next to rest the most commonly

useful treatment is the application of heat to the limb; this may be done in various ways, as by the application of hot bricks, sand-bags wrapped in flannel, linseed and mustard or antiphlogistine poultices. If mustard is used it should not be in greater strength than one part in twelve of linseed, as for continuous application the use of mustard in greater strength is liable to blister the skin. Radiant heat by the employment of large carbon filament lamps is often very comforting and may induce sleep.

This form of heat may be applied either locally to the buttock and thigh by means of a couple of large lamps attached to a curved reflector, or the patient may be given a so-called radiant heat bath by being surrounded on a special couch or frame with these lamps. Another very valuable form of electric heat is diathermy, in which the oscillating current of a modified form of high-frequency apparatus is applied by means of moist pads to the thigh and buttock, and gives penetrating heat unobtainable by other means. High-frequency currents in the form of so-called violet rays are often employed but have little other than suggestive value. Ultra-violet rays from a quartz mercury-vapour lamp, or from a tungsten arc, may give good results.

Electricity in the form of constant current, or *ionisation*, is sometimes extremely valuable; it is best applied twice daily for about twenty minutes by means of large electrodes thickly padded by wetted Gamgee tissue or layers of lint, one large pad attached to the negative wire being applied over the buttock, wetted with one per cent. salicylate of soda, and the other electrode, about twelve by eight inches in size, bent round the under surface of the thigh just above the knee, wetted with lithium carbonate, and attached to the positive wire. Using a constant current of from twenty to forty milliamperes, the salicyl radicle enters the tissues at the negative electrode, while lithium is absorbed from the positive electrode. Special care must be taken when daily ionisation is employed to avoid damaging the skin by electrolysis. This will occur very rapidly if bare metal

or wire be allowed to press upon the skin during the treatment, as blisters are easily raised at such points, and electric burns of this nature are very stubborn and heal very slowly. Extra thick moist padding of the electrodes is a useful safeguard, but even with the greatest care daily ionisation is apt to produce crops of small pimples underneath the electrodes, which will break down into small ulcers unless the treatment is intermitted. X-ray treatment over the nerve may then be used instead with advantage. In some cases electricity increases the pain, or in other ways is an irritation and annoyance to the patient, so that it must be conceded that some persons cannot bear electrical treatment in any shape or form.

For them blisters may be applied to the skin along the course of the nerve and may be repeated twice weekly, or as an alternative ABC liniment may be rubbed gently twice daily on the buttock and back of the thigh. Painting with liniment of iodine or strong hydrochloric acid down the back of the thigh has also been practised with success. Massage must be avoided in the early stages of an acute sciatica, though later on, after the lapse of six or eight weeks, light massage may be begun, combined with passive movements and Swedish exercises, the nerve being gradually stretched by passive movements of extension.

Sources of sepsis must be looked for, such as antral or frontal sinusitis, and carious teeth and pyorrhœa should be dealt with. Septic absorption of intestinal origin may be treated with auto-vaccines if cultures of the stools indicate this line of attack.

Medicinally, iodide of potash and salicylates should be tried, and slightly purgative doses of sodium sulphate daily sometimes succeed alone in obstinate cases. As in brachial neuritis, analgesics to control the pain are usually necessary, and combinations of pyramidon, aspirin, medinal and codeine or heroin may be used to relieve the pain for a few hours at a time and to induce sleep. Injections of morphine, indeed, may be required at times, while the addition of hyoscine hydrobromide gr. 1/150th

ter in die to the mixture may be useful in neurasthenic subjects.

In many cases of sciatica the Achilles jerk is lost after the first few days, and I have seen in cases of bi-lateral sciatica the Achilles jerk return as soon as the pain left one limb, with disappearance of the other Achilles jerk as soon as the pain attacked the remaining limb. Indeed the loss of an Achilles jerk is a useful proof of a neuritis of the sciatic nerve, and may be a valuable point in differentiating this pain from other neuralgias of the limb, such as arthritis of the hip-joint or anterior crural neuritis. In the latter disease it is the knee jerk that will be lost and not the Achilles jerk. In old-standing cases of chronic sciatica further evidences of damage to the nerve trunk may be seen, such as coldness and blueness of the foot, anæsthesia and muscular wasting below the knee, and in a few such cases the pain is resistant to all treatment except mechanical scarification of the nerve sheath.

For this the nerve must be exposed by an open operation, and it may be found shrunken into a round, fibrous-looking cord, the size of the little finger. Mechanical stretching of the nerve longitudinally by such open operation and forcible traction upon the nerve itself is usually of no benefit, and should, I think, be avoided.

Saline Injections in Sciatica

Cases in which the pain is persistent and improvement is delayed after four weeks may often be treated most successfully by injection of the nerve trunk with fifty to eighty cubic centimetres of normal saline solution.

This treatment will be more especially indicated when there is tenderness to pressure on the buttock over the sciatic notch, or between the tuber ischii and the lesser trochanter, and when the pain is aggravated by extension of the nerve by flexing the hip with the knee straight.

The method of injecting the nerve is to use a hollow needle of four or even five inches in length and a ten cubic centi-

metre Record syringe. The course of the nerve is mapped out on the skin and the notch marked.

This is roughly four inches external to the median line at the top of the intergluteal cleft and four inches above the tuber ischii. From this point the nerve passes outwards and downwards between the tuber ischii and lesser trochanter. This line is drawn on the skin, and a point on the line one and a half inches below the notch is marked. The skin at this point is then injected with two per cent. novocain solution, and the long needle is then taken and passed directly down to the notch. Usually the ilium will be felt at a depth of about three inches, and the nerve must be searched for until found. This is indicated by the patient feeling an electric thrill down the leg into the foot or heel. When the patient announces this the needle is held quite steadily and one cubic centimetre of saline injected. If this gives pain in the foot then the point of the needle is correctly placed within the nerve and two cubic centimetres of sterile two per cent. novocain solution are then slowly injected. If the needle point has remained within the nerve, the foot soon becomes numbed and the pain ceases. The needle is held still in position and a larger syringe filled with warm freshly-boiled normal saline is fitted on and the contents of the syringe are slowly injected. In this way fifty to eighty cubic centimetres may be injected, and the limb is usually perfectly comfortable afterwards. If the preliminary small saline injection causes no sensation in the foot then the point has either passed through or to one side of the nerve and must be readjusted.

In some cases there may be after injection some discomfort and feverishness with loss of sleep the first night, best controlled by a hypodermic injection of a quarter of a grain of morphine. The patient must be kept in bed for twelve hours at least, and better still for three or four days.

In most cases a second injection is advisable on the fifth day, when I usually inject the nerve at the level of the tuber ischii, thus including the upper four inches of the

nerve between the notch and the tuber ischii in the two injections, which is the area of the nerve most usually involved by the perineuritis.

The effect of the saline injection into the nerve is to cause it to swell up in an egg-shaped form, separating the nerve bundles and bursting asunder laterally any adhesions that have been formed. Quite a large number of sciaticas that are threatening to become chronic may be relieved and even cured by this treatment.

It should be needless to say that alcohol must never be used for injection of the nerve trunk in sciatica, on account of the foot-drop and paralysis of all the muscles below the knee that would ensue.

Another injection treatment that has been employed frequently on the Continent is injection of the **sacro-coccygeal foramen** with novocain and alcohol. This foramen in the median line is not difficult to reach, and the lower sacral roots of the cauda equina above are shut off from the general intrathecal space above, so that the injection remains local in its effects.

Perhaps the type of sciatica in which saline injection of the nerve in the buttock is the most immediately successful is that which follows injury from a fall on the buttock or from sudden violent muscular action, causing rupture of some muscular fibres with immediate pain in the buttock and leg. In nearly all such cases the damage is located between the sciatic notch and the tuber ischii, and one or two injections of fifty to eighty cubic centimetres of saline into the nerve at these two points, as above described, may produce an immediate or even a dramatic cure.

Scoliotic Sciatica

Many cases of sciatica after persisting for several weeks develop lateral flexion of the spine, or scoliosis. There are two types, the homo-lateral and the contra-lateral scoliosis. In the former, the body is apparently drawn down on the affected side by contracture of the erector spinæ on the same side. Though this type may persist for two or three years

if proper rest and treatment are not adopted, it is usually much more amenable to rest and to saline injection treatment than is the contra-lateral type. Two injections of sixty to eighty cubic centimetres of warm sterile saline at an interval of four or five days, placed properly within the nerve at the sciatic notch, and at the level of the tuber ischii, with continual rest in bed, will usually relieve the contracture, and the patient may rapidly get well. In these cases the contracture is protective, to prevent extension of the nerve, and is an indication of the perineuritis affecting the nerve chiefly outside the pelvis.

Contra-lateral scoliotic sciatica is nearly always more chronic and difficult to cure. The spine is flexed laterally away from the affected side, and the scoliosis in this type is probably due to the fibrositis involving chiefly the fourth and fifth posterior divisions of the lumbar nerves close to the intervertebral foramina. Lateral flexion of the spine to the same side compresses the inflamed tissue near the vertebræ, hence causing pain along the fourth lumbar nerve. The fifth lumbar nerve would be less affected by such lateral flexion, and the first sacral nerve, issuing from the rigid sacrum, would, of course, not be affected at all. The scoliosis or lateral flexion of the spine away from the affected side is therefore protective in this type also, and is due to a tonic contracture of the erector spinæ on the opposite side pulling the spine over, so that when the patient stands as erect as he can a plumb line dropped from the seventh cervical spine may pass as much as five inches to the opposite side of the gluteal cleft. The patient is conscious of this deformity, and usually ascribes it to leaning his weight constantly on the sound leg. It is, however, a protective contracture, and if the spine is forcibly straightened while the patient is standing, by pulling the shoulders towards the affected side while the knee is held against the patient's hip so as to give good leverage, pain is at once set up down the thigh and leg to the ankle.

Moreover, if the patient is laid flat upon the floor with his arms by his side and encouraged to relax all his muscles,

it is then easy to straighten the spine so that all the scoliosis disappears, without causing any pain, though often now the patient will say that he feels that he is bent over to one side. When standing it may easily be shown that there is no tilting of the pelvis, but that all the scoliosis takes place above the lumbo-sacral joint.

FIGS. 26 and 27.—Contra-lateral Scoliotic Sciatica, right leg.

In this type of contra-lateral scoliotic sciatica the Achilles jerk is usually unaffected, which is indeed no matter of surprise if, as I suggest, it is chiefly the fourth lumbar nerve that is involved in these cases, the Achilles jerk being served by the posterior tibial portion of the sciatic nerve, which is derived from the first and second sacral nerves.

I have always found that this type of sciatica is not benefited by saline injections of the nerve trunk, and this difference of reaction to the treatment, as compared with the homo-lateral scoliotic type, is easily understood if in the former type the site of the fibrositis is close to the intervertebral foramina, and thus out of reach of saline injections into the nerve after its exit from the pelvis at the sciatic notch.

In many of the scoliotic sciaticas of both types tenderness is present on deep palpation between the sciatic notch and the fourth lumbar transverse process, a common site for the tenderness being in the region of the posterior superior iliac spine, which can usually be felt by palpation, even in the fattest subjects, and of the posterior first sacral foramen, which lies about half an inch internally and above this point.

Sometimes there is present in the contra-lateral scoliotic type a spot of intense tenderness on deep pressure over the site of the fourth lumbar intervertebral foramen, an inch outside the middle of the fourth lumbar spine. Deep alcohol puncture of these tender points sometimes gives immediate good results, and I saw in 1915 immediate and complete cure follow such puncture of the region of the fourth lumbar foramen in an officer after the pain and scoliosis had lasted for months. This method of injection to the intervertebral foramina is now known as *para-vertebral* injection, and has been used for various chronic spinal neuralgias, both dorsal and lumbar, such as the pain in the chest in aortitis and chronic pleurisy.

Sciatica with Arthritis of Hip

Sciatic pain of the usual distribution down the back of the thigh and leg into the ankle is not at all a rare accompaniment of chronic rheumatoid arthritis of the hip-joint. The probable causation of such sciatica is a perineuritis due to direct spread of fibrositic inflammation from the hip-joint backwards to the neighbouring sciatic nerve. The proof that the sciatic nerve is truly involved, and that

the pain is not due to the arthritis alone will be loss of the Achilles jerk, with pain on extension of the nerve, though the same amount of flexion of the hip when the knee is kept flexed does not produce the pain. A very common mistake is to overlook arthritis of the hip-joint when the patient is complaining of pain around the hip and buttock and thigh down to the knee. In all such cases careful examination of the hip-joint is necessary before considering the diagnosis of sciatica as an explanation of the pain, but it must be remembered that in a certain number of cases there is a doubly acting source of pain, namely, chronic arthritis of the hip plus sciatic perineuritis. The treatment of such an additional sciatica will be on the ordinary lines, but it is usually more chronic and resistant to remedial measures.

Osteo-arthritis of the Hip-joint

Even more liable to misinterpretation is the varying pain below the knee on the outer side of the ankle, that may continue for several years in early osteo-arthritis of the hip-joint, without any pain being felt in the hip, thigh or knee. It is usually aggravated by long-continued standing or walking, as in shooting, and a bad attack may thus be attributed to a new pair of boots or gaiters, to a blow or to any cause rather than to the hip-joint. The gait in such cases will usually suggest hip-joint disease, and there will be difficulty in turning rapidly, owing to increased pain in the ankle.

The hip-joint should always be most carefully examined in such cases of chronic pain in the leg, and limitation of rotation or abduction noted, while a good X-ray photograph of the joint will demonstrate the flattening of the head of the femur, leading to a mushroom appearance, with osteophytic outgrowths around the edge of the acetabulum.

CHAPTER VI

SACRALGIA

Sacralgia and other Psychalgias; Coccygodynia

Vague pains around the sacrum, sometimes spreading below to the coccyx, or around the hip and even down to the heel, simulating a sciatica, may torment persons of nervous heredity for years, or even for most of their lives.

In women, the site of the pain is more often sacral or lumbar, sometimes at various points with local tenderness close to the vertebral spines up to the dorsal region. Occasionally coccygodynia in women may follow childbirth or a fall in the sitting posture, and pain and tenderness in this region may at first be due directly to trauma, though its continuance for years is usually to be ascribed to superadded neurosis which stereotypes the pain, long after the real local effects of the injury have disappeared. Men of neurotic taint may also suffer from coccygodynia, and in them the pain is liable to spread to the end of the penis, or into one testicle, or into the groin and down one leg.

A clergyman *æt.* 63 complained of sensations of discomfort always present from the coccyx to the end of the penis, resembling the sensation of "bearing down," and aggravated by the sitting posture. As a boy he had fallen backwards in a sitting posture on a stone seat. Three years later pain and discomfort started in the region of the coccyx. He was always delicate and very neurotic; one brother and two aunts had been insane, and he himself was impotent on marriage. The coccyx was removed by a surgeon without apparent benefit. This type of case is a true psychalgia, though the jarring of the whole nervous system by the fall and the local bruising determine the site of the subsequent neuralgia.

Again, a neurotic man, who had had a nervous breakdown, married at 26. For several weeks he was unable to penetrate, and within three months pain suddenly began one morning in the left testicle, spreading along the groin to the back, into the left arm and down the front of the left thigh into the heel. When I saw him ten years later, he complained of pain all day long on the left side, with shooting pain in the testicle. Any sudden shock would cause pain in the groin and stomach. The left testicle was excessively hyperæsthetic, and he constantly wore a suspender, the scrotum also being very sensitive, and slight pressure on the testicle caused a sinking sensation with dejection and sobbing. He was liable to attacks of intense depression (see Visceral Neuralgias, p. 383).

A man. *æt.* 52, much worried during the War with overwork, had for three years complained of pain above the coccyx and at the back of the left hip, the whole leg aching down to the heel. The pain spread to the bladder region, producing polyuria. At the commencement of the neuralgia the pain was situated between the legs, before it settled into the coccyx region, and he said that at first he must have imagined much of it. Local treatment by diathermy and massage produced temporary great improvement, but the symptoms soon returned as acutely as ever.

Sacralgia is sometimes accompanied by a varying amount of swelling over the sacrum, resembling a firm œdema, but not pitting on pressure. At times this swelling is scarcely noticeable, and at others the swelling may be sufficiently obvious to attract undue attention, and may even trap the unwary into a diagnosis of cellulitis, sacral caries, etc.

The pain is usually continuous, though variable in severity, and may spread to the groins and thighs, and sometimes a clue to the psychical nature of the pain may be found in the obvious obsession of the patient as to the intensity of the pain, a continual complaint being made of the pain being unbearable. On examination there is usually tenderness, and sometimes intense hyperæthesia of the sacral region, though the functional nature of this

tenderness may be demonstrated by distracting the patient's attention towards another part of the spine which is being examined, while firm pressure gradually applied upon the previous intensely hyperæsthetic sacrum may attract no notice, and no pain is complained of until the attention is again directed to this part.

In some women similar pain is complained of across the lumbar region, or there may be an area of tenderness succeeding a blow upon the back many years before.

A woman, *æt.* 28, when riding pillion with her husband on a motor-cycle, was thrown and bruised her back. Pain in the back near the twelfth dorsal spine worried her constantly for the next twelve years, so that she was unable properly to look after her home and children. No abnormal physical sign could be detected, the spine was quite mobile, but the spinal column was intensely tender to pressure at and near the twelfth dorsal spine. One application of the cautery cured her, and she has since been perfectly free of pain and able to do her work.

Genital Neuralgia, a form of psychalgia in which a fixed pain affects the genital organs, is much commoner in males than in females. Pain running into the testicle has already been referred to, and occasionally a chronic fixed pain may affect the penis. As in the other psychalgias, persons of neurotic family or racial heredity are more prone to suffer. An unmarried Jew, *æt.* 34, had for five years suffered with continuous pain referred to the tip of the penis and along the frænum, appearing to start from an old circumcision scar. His sleep was much disturbed by means of it, and he was unable to distract his thoughts from the pain for long. X-ray examination for stone proved negative, and no abnormal physical signs could be found, and he complained that he had spent all his money on this trouble without getting relief. A year or two later the pain disappeared for no apparent reason.

Sexual Psychalgia.—Neuralgic pains around the loins and spreading into the lower limbs is sometimes met with in women from sexual irritability and unsatisfied sexual

emotions. A lady of 43, married to a much older and frigid husband, had complained for six years of pain across the loins like toothache, spreading down the right leg, even to the ankle. Her pain was apt to be intermittent, and like many neuralgias was much influenced by emotion. Three weeks before I saw her she had seen a child run over and badly injured by a motor-car, and ten minutes later she felt acute pains in the loins and right leg, and she was brought to me as a case of sciatica. Treatment on ordinary lines effected no improvement, but a doctor brother who had her confidence gave the right clue, and psychotherapy and suggestion treatment cured her in a few weeks.

In unmarried girls similar neuralgias due to the above cause are occasionally met with, tender points simulating fibrositis, and apt to shift position after local treatment such as the cautery or alcohol piqure. In one case inability to walk far or to play games is constantly professed, though in another the pain may be absent while she is able to dance several nights in succession. A feature of this psychalgia is that it is often severe or even worse at night when in bed. The diagnosis of these cases is sometimes very difficult, though the patient's manner, especially while being examined, may give the necessary clue, and it behoves the medical man particularly to be on his guard while dealing with such patients.

Psychalgias of sexual origin are certainly not limited to the lumbo-sacral region, but it is especially the type above described that calls for more than ordinary caution, and vaginal examination should be dispensed with unless absolutely necessary, and then undertaken only in the presence of a nurse or relative of the patient.

Psychalgias may afflict any part of the body, and being of mental origin the pain is unlikely to correspond closely with any anatomical area of nerve root distribution, but is more likely to be segmental, involving a toe or finger, the tongue or one side of the face. There is usually a clue to be obtained from a history of previous mental breakdowns, or a family history of insanity or other neuroses.

A clergyman aged 63 had suffered from pain in his right great toe for two and a half years, for which he had consulted many surgeons and physicians, and had suffered much at their hands, including an operation to remove a supposed exostosis, tenotomy, and excision of the metatarsophalangeal joint of the great toe. Electrical treatment was tried by one, and another diagnosed a kink in his colon, but all was of no avail and the pain grew much worse. His wife had noticed how his pain disappeared at once when he was thoroughly interested, and as he was the son of first cousins, with a strong taint of insanity in the family, the diagnosis of psychalgia seemed more than probable.

Psychalgia may simulate in its distribution a trigeminal neuralgia, though the pain is never of the paroxysmal type evoked by eating or by handling the face that is so characteristic of the true neuralgia major of the fifth nerve. A good object lesson of facial psychalgia was the case of a Jew *æt.* 44, who had complained for nine months of pricking pains on both sides of his nose, especially on the left side. As he had formerly had syphilis, full treatment by mercury and iodide and arsenobenzol injections was carried out, but after another year the pain had increased to a constant aching and spasmodic burning pain in the left cheek and nose.

Alcohol injection of the second division of the fifth nerve at the foramen rotundum produced dense anæsthesia of the cheek and palate, but three weeks later he complained of the pain being most agonising, no drugs giving any relief. Two months later alcohol injection of the Gasserian ganglion was performed, total trigeminal anæsthesia resulting, which was as complete and lasting as after a successful gasserectomy operation. The pain nevertheless persisted apparently unaltered, and his mental condition became exceedingly neurasthenic. A year later he went to a well-known surgeon for gasserectomy to be done, but this was refused after the permanence of the trigeminal anæsthesia was observed and his mental state noted. He found another surgeon a few months afterwards who operated on the Gasserian ganglion, but the pain remained as bad as ever.

One of the commonest types of psychalgia affecting the head is the well-known vertical pressure pain, like a weight pressing down on the top of the head. Sometimes this is varied by a sensation as of opening and shutting of the top of the head. This pain is sometimes described as "clavus hystericus." Headache of this nature should not lead a careful physician into the error of mistaking it for the headache of cerebral tumour, but I once fell into this error when a famous ophthalmic surgeon had reported the presence of optic neuritis. This appearance of optic neuritis proved to be due to the presence of high myopia, which may cause an appearance of the disc and the surrounding retina superficially resembling papillœdema.

This pressure pain on the vertex or crown is usually worse in the morning, as is characteristic of neurasthenic depression, and may be accompanied by irregular tic-like contractions of the occipito-frontalis and other facial muscles during the greater part of the day.

The treatment of the psychalgias depends largely on the personal ascendancy that the physician is able to establish over his patient, and this may be aided by local strong faradic treatment, or sometimes the actual cautery to the skin in certain cases, as in sacralgia and similar psychalgias. Operative treatment, however, including alcohol injections of nerve trunks, should be avoided, as once this has been done a physical basis for the pain has been established, and it will be far more difficult if not impossible to dislodge the mental attitude from this stronghold by suggestion, psychanalysis or other forms of mental treatment.

The osteopath and the manipulative surgeon too will usually fail also, as their appeal is to the presence of a local trauma, and not to the inhibition of a disordered mental process. Indeed, the most hopeful course sometimes is to supplement the form of insanity afflicting the patient by another, by conversion to Christian Science, or a visit to Lourdes may effect the mental cure that is necessary, when orthodox medicine has failed.

CHAPTER VII

NEURALGIAS OF THE HEAD AND NECK

The term neuralgia, meaning a pain referred along a nerve distribution, may in practice be distinguished from the term neuritis in that neuralgia indicates pain referred often widely along many nerve branches from a single small focus of disease affecting a few nerve filaments, while the term neuritis will include lesions of nerve trunks or small nerve branches.

Dental Neuralgia.—For example, dental neuralgia due to caries of a tooth producing inflammation of the pulp cavity, or acute pulpitis, may itself cause tenderness and pain referred only to that tooth, that is to say, neuritis of the dental nerve filament of that tooth. Soon, however, in many cases the pain begins to radiate beyond the affected tooth along the lower or upper jaw as the case may be, or indeed the pain may be referred to the upper jaw only when the offending tooth is in the lower jaw, and *vice versa*. It should be noted, however, that dental neuralgia is never reflected across the middle line, a fact which appears to be insufficiently appreciated by practitioners, if I may judge from the number of cases in which I have seen all the teeth on both sides removed in the effort to arrest a chronic trigeminal neuralgia. Dental neuralgia may affect areas beyond the upper or the lower jaw, and sometimes the pain will spread over the whole fifth nerve territory and extend down into the neck and arm, even to the fingers. Such a wide distribution as this is rare, and is more likely to be met with in subjects of a specially nervous temperament. The different teeth have each their special area of referred pain, most notable perhaps being the lower molars,

especially the wisdom, disease of which so often causes pain referred into the ear and down the side of the neck.

The lower wisdom is a tooth specially likely to give trouble: firstly, the tips of its two roots are in close proximity to the inferior dental nerve, and may even enclose this nerve between them. Violent neuralgia may be produced in the young adult by the erupting wisdom tooth encountering toughened gum over it, but incision of the swollen purplish gum over the tooth will at once relieve the pain. A not uncommon developmental defect in the position of the lower wisdom tooth is due probably to the shortening of the mandible which is taking place in the human race, a result in all probability of the extensive use of soft cooked foods and consequently less and less use of the jaw for biting and grinding purposes. The last molar thus gets tilted forwards, its roots lying against the base of the coronoid process and the crown of the tooth lying against the side of the second molar in front. In many cases this may lead to no damage being done and the tooth may remain in this position throughout life without ever erupting or causing any disturbance. In some instances after puberty severe pain may be caused by the pressure of the wisdom forwards against the second molar, and the appearance of severe intractable neuralgia referred along the lower jaw and into the ear in a young adult should always lead to careful examination of the teeth. If the wisdom has never erupted, X-ray films should be taken, and if the lower molar seems to be impacted in the position described, it will probably be advisable to remove either it or the second molar in front of it.

Usually the latter procedure is chosen, owing to the considerable difficulty of extracting the buried impacted wisdom. In quite a number of individuals, however, complaining of neuralgia of the jaw, X-ray films may reveal a total absence of all four wisdom teeth.

Dental neuralgia is often classed as one of the minor neuralgias of the fifth nerve, though the severity of the

pain produced by this cause may be sufficient to provoke attempts at suicide, as I have known in two cases.

In the investigation of a facial neuralgia it is very important to examine the teeth carefully and to take no one else's word for their being sound, as dental causes are far and away the commonest origin of facial pain. The teeth should be carefully examined, and a dental or laryngeal mirror used for the inspection of their posterior surfaces. The teeth should be tapped with a light metal instrument and syringing of the individual teeth with hot and cold water alternately may indicate a local periostitis. I have known a severe and intractable neuralgia of the lower jaw due to a tiny hole leading into the pulp cavity of a molar tooth, into which the bristle of a toothbrush had become firmly wedged. In another case I was asked to inject the fifth nerve for trigeminal neuralgia for severe pain which had lasted ten days in a man of over 50. The pain was so severe that he said if nothing could be done to relieve it he would shoot himself. On inquiring about the teeth both the patient and the doctor declared that he had none at all, for he was wearing two complete dentures. Unwisely I accepted their statements without having the dentures out to look for myself, and I injected the foramen ovale, and relieved his pain. He still complained, however, of slight grumbling pain referred to the lower jaw, which convinced me that there must be some dental cause persisting. On having the lower denture taken out the source of his neuralgia was at once apparent. The right lower canine tooth had been left behind for the support of his plate, and the neck of this tooth had been half sawn through by the hook attachment of the plate. In cases of recent development of neuralgia no diagnosis of chronic intractable trigeminal neuralgia should be made until full examination of the teeth and jaws on that side, including X-ray films, has been made.

One of the commonest sources of dental neuralgia is caries of a tooth, the cavity being irritated by sweet and decomposing food. A useful remedy for such pain, which

might with advantage be kept in every household, is a solution of equal parts of carbolic acid, menthol, and oil of cloves. A small bottle containing two drachms of this mixture may be kept for such emergencies, and a very small piece of cotton wool soaked in this oily liquid may be pressed into the cavity with the point of a wooden match, or better still fine forceps, after the cavity has been cleaned from food detritus as far as possible. The relief from pain is almost immediate, but the carious tooth should be attended to by a dentist as soon after as may be practicable.

Sinusitis.—Inflammation of the lining membrane of the cavities in the facial bones is often a source of paroxysmal neuralgia and recurrent headaches. Thus, antral abscess, catarrh and suppuration in the frontal sinuses are common sources of facial pain and recurrent headaches. Antral abscess is not uncommonly secondary to dental caries, but, like infection of the frontal, ethmoidal and sphenoidal sinuses, it is more usually produced by catarrh and inflammation of the nasal passages. Both in frontal sinus and antral inflammation I have frequently seen marked periodicity in the neuralgic pain, which usually comes on at ten or eleven in the morning and lasts until about five p.m. There is generally some tenderness on pressure over the sinus involved, and probably a history of recent and present discharge of muco-pus from the nostril on that side.

Antral pain at the commencement is referred over the second division of the fifth, but I have seen it spread over the whole trigeminal area, and even down the neck and arm. Two methods of diagnosis of antral and frontal sinus accumulations which are never to be neglected are: first, transillumination, secondly, X-rays. The former is easily carried out in the consulting-room, if the room can be adequately darkened, a small electric bulb, such as the lamp of an electric ophthalmoscope, being necessary, and held in the mouth with the lips closed. Any movable dentures must, of course, first be removed. By this means the amount of

illumination of the two antral sinuses can be compared, and if one contains an accumulation of muco-pus, its dark shadow will contrast strongly with the brightness on the other side. For the examination of the frontal sinus the small electric bulb is pressed into the orbit over the upper eyelid as far as can comfortably be borne, and a normal frontal sinus will give a bright red glow up to the middle line above the superciliary ridge; this process is then repeated on the other side and the two sides can be compared. X-ray photographs should also be taken laterally and antero-posteriorly. In the latter position the frontal sinuses and antral sinuses will show up best, while in the lateral view the sphenoidal sinus shows best immediately in front of the sella turcica, from which it is separated by a comparatively thin sheet of bone. In the lateral view also the jaws and teeth will be well seen and also the ethmoidal sinuses.

Syringing out the antrum or frontal sinus is an operation which belongs more particularly to the throat and nose specialist, and may be curative as well as diagnostic of accumulations of pus and mucus in those cavities.

The openings into them have usually to be enlarged, but all that is necessary can be done from within the nose or cheek without cutting the skin externally.

The type of neuralgia due to muco-purulent accumulation in the ethmoidal sinus has been described by Sluder, in which the pain is referred along the roof of the mouth and back of the nose and eye extending into the cheek.

This is ascribed to irritation of the spheno-palatine or Meckel's ganglion, as it lies close to the spheno-palatine foramen.

Paroxysmal Supra-orbital Neuralgia.—In frontal sinus suppuration the pain is mostly distributed along the course of this nerve, tenderness usually being present on pressure over the supra-orbital notch. The pain is rarely constant, and is often periodic daily from morning until late afternoon, and a history of influenza or other recent nasal catarrh may be obtained. I have met some

cases in which the onset of the neuralgia may directly be attributed to the too forcible inhalation of nasal antiseptics, thus directly causing infection of the frontal sinus. The pain is sometimes neuralgic, and sometimes more headachy in character, and its origin is easily overlooked, and the diagnosis mistaken for migrainous neuralgia and other forms, unless transillumination and X-ray examinations as above described are undertaken. There are, however, other forms of periodic supra-orbital neuralgia, in which the sinuses are perfectly innocent. Malaria has the credit of producing this condition, though it must be very rare in this country. Influenza certainly may be followed by this condition, without any definite suppuration in the frontal sinus, and the attacks may pass away without any operative form of treatment. It is arguable whether the pain in this case is a toxic perineuritis of the supra-orbital nerve, comparable to a post-febrile sciatica, or whether it is dependent upon some congestion and temporary blockage of the secretion within the frontal sinus.

A chronic form of recurrent periodic supra-orbital neuralgia is sometimes met with in which the pain is definitely neuralgic along the course of the nerve and lasting for many years. This form may follow slight injury to the forehead, such as a fall, and is probably due to involvement of the supra-orbital nerve in fibrous tissue, or the nerve may be gradually pressed on by inclusion in a bony canal at the supra-orbital notch. This cause of frontal neuralgia may be compared to the pain along the ulnar nerve that may result from similar pressure upon the ulnar nerve in the condylar groove. The best treatment for this form of long-standing supra-orbital neuralgia is excision of the nerve below the notch as it runs along the roof of the orbit.

Another type of periodic supra-orbital neuralgia is probably *migrainous* in origin (see p. 299); the pain is referred to the back of the eye or even into the eyeball, and in a less degree to the frontal area and the upper part of the cheek. The pain is usually present in

slight degree on waking at six or seven a.m. and increases rapidly in severity to an almost insupportable degree, rendering the patient quite incapable of work or attending to business for several hours. During the afternoon the pain wears off and he may be quite free from the neuralgia in the evening.

There is rarely any vomiting with this type of headache, though slight nausea is sometimes present. Next day duplicates the horrors of yesterday, and repeatedly for a week or ten days the sufferer is in daily torment until the afternoon or evening. The later attacks will be shorter and less severe, and the patient will then be free from them completely for an interval which varies from weeks to months. I have seen migrainous neuralgia in this fashion attack a man of 50 day by day for six weeks every year in the autumn, waking him up at three a.m. with piercing pain in the temple, which gradually grew in severity as above described. These attacks had lasted for very many years.

Treatment of Supra-orbital Neuralgia

The treatment of these cases of recurrent frontal neuralgia is difficult.

Once the pain has started, the attack almost invariably pursues its course unchecked by any analgesic draught or powder. I have tried, and others also, alcohol injection of the supra-orbital notch, with only occasional good result, and I have also injected the infra-orbital nerve for paroxysmal pain in the eyeball, twice with complete success, no pain recurring for three years. The only really satisfactory way of cutting short the attacks which I have been able to devise is to give daily anticipatory injections of morphine and hyoscine; for example, if the patient usually wakes at six o'clock with pain, I arrange to have him given at four a.m. a hypodermic injection of one-quarter grain of morphine with one-hundred-and-fiftieth of a grain of scopolamine. As a rule, this aborts the attack both for duration and severity. The patient should stay in bed

till mid-day, and he may then get up for the afternoon and evening.

Three or four injections repeated early every morning will usually break the intensity of the attack, the pain then subsiding and becoming shorter and less severe each day.

As has already been described, supra-orbital neuralgia may be a form of migrainous pain, and in those cases in which the neuralgia is limited mainly to the territory of that nerve, alcohol injection of the nerve at the supra-orbital notch may be completely successful. There is, however, no certainty in the treatment of this type of neuralgia, such as can be practically guaranteed in paroxysmal tic douloureux. Sudden onset of violent supra-orbital neuralgia is also common in association with chills, bad cold in the head and influenza. In some of these cases the pain is undoubtedly reflex from infection and accumulation of muco-pus in the frontal sinus of one side, and transillumination or X-rays may demonstrate an opaque sinus. The pain in these cases is often regularly periodic, starting at ten or eleven in the morning and disappearing about five in the afternoon. Tenderness is usually present over the supra-orbital notch, and there may be hyperæsthesia of the forehead to a light scratch.

In others, however, the frontal sinus may be found quite normal, with violent supra-orbital neuralgia occurring daily for several hours in the morning. Complete relief may then be obtained in these cases by the injection of the nerve at the supra-orbital notch by alcohol, preceded by novocain.

An interesting case of supra-orbital neuralgia started in a girl aged 10, who was said to have had frontal herpes at that time. When I saw her 17 years afterwards she had suffered ever since with right supra-orbital neuralgia, the pain being of a grinding character and aggravated by stooping or by excitement, or by hot, stuffy rooms. The attacks of pain were periodic, but no scars of herpes were visible, nor was there any anæsthesia. As alcohol injection by another physician had failed to give relief, I recom-

mended excision of the supra-orbital nerve, which was done, the result being completely successful and permanent.

Occipital Neuralgia

Occipital neuralgia may result from a peri-neuritis or so-called rheumatic inflammation of the nerve sheath following exposure to cold of the neck and back of the head. The pain spreads up the back of the neck and head behind the ear on one side to the top of the head, and is liable to occur in paroxysms which may last as long as an hour, or may occur as often as every three hours. The pain is apt to get worse when warm in bed, though it may be directly due to chill, an apparent contradiction which is often seen with other rheumatic pains. This condition may be set up by the draught affecting the back of the head and neck when driving in a car or from an open window in a railway carriage.

I have known it start in a woman of 50 who while telephoning in a very cold room noticed a cold wave appear to strike the left side of her head and face. The same evening she began to suffer from occipital pain on the left side, which soon developed into severe spasms occurring every three hours. Aspirin would relieve the pain for the time, but the severity of the attacks continued for five weeks until alcohol injection of the nerve was done.

In these cases there is almost invariably a tenderness over the site of the nerve as it passes over the occipital bone, and it is a useful treatment to attempt injection of the nerve by passing a hypodermic needle vertically downwards to the bone about half an inch from the middle line and about half an inch below the level of the occipital protuberance.

Two per cent. novocain should first be injected, and after waiting about one minute the syringe should be changed without moving the needle and about 7 m. of 90 per cent. alcohol injected. It is highly probable that the actual nerve fibres may not be reached by the alcohol, and anæsthesia of its nerve distribution on the back of the

head usually does not result; however, the perineuritic injection of alcohol appears to be sufficient in most cases to give relief.

The type of this occipital pain and its reaction to alcohol injection agrees closely with some of the periodic supra-orbital neuralgias which may follow influenza or colds in the head without any evidence in some of them of frontal sinus distension or abscess. In some of these, alcohol injections of the supra-orbital nerve at the notch give instantaneous relief and the pain may cease. Occipital neuralgia may also be associated with neuro-fibrositis of the shoulder and neck muscles in which the neuralgia is then a referred pain and is not due to a peri-neuritis of the great occipital nerve itself. In such cases there will be no local tenderness over the course of the nerve on the occipital bone.

Neuralgia will be complained of radiating over the shoulder and into the neck as well as up the back of the head, and the pain will probably be aggravated by movements of the shoulder. Local spots of intense tenderness will probably be found in the region of the supra-scapular fossa or internal to the upper portion of the vertebral border of the scapula. Careful alcohol injection of these tender points may produce a brilliant cure of chronic pains which have lasted for years (see Neuro-fibrositis). In addition to these forms of fibrous rheumatism, which often develop insidiously and without any apparent cause, occipital neuralgia may be due to osteo-arthritis between the atlas and axis vertebræ, or to vertebral caries. Being derived from the posterior branch of the second cervical nerve, osteophytes at the intervertebral foramen may cause persistent and troublesome neuralgia. Secondary carcinomatous deposits in the bone and new growths in the membranes and upper part of the spinal cord may also be the source of referred pains in this region.

CHAPTER VIII

CHRONIC PAROXYSMAL TRIGEMINAL NEURALGIA

Alternative names for this disease are tic douloureux, epileptiform neuralgia, prosopalgia, trifacial neuralgia, neuralgia major, or Fothergill's Disease. This peculiarly violent and intractable neuralgia of the face has probably been known since civilisation began, though the first connected account of the disease seems to be that of the elder Fothergill, who published his monograph on "A Painful Affection of the Face" in 1773.

In the course of a large practice he seems to have met with some fourteen cases, and on account of the persistence and incurability of the pain he attributed the cause to some form of cancer.

Visitors to Wells Cathedral in Somerset may notice some of the carved capitals on the two pillars facing the south aisle of the choir within the cathedral, three of which at least are illustrations of persons suffering violent facial pain. One of a monk in his cowl with wide-open mouth, and tongue showing, with features indicating severe agony, is a typical portrait of a spasm of tic douloureux. These carved capitals bear silent and immemorial witness to the thank-offerings given to the cathedral funds by pilgrims to the tomb of Bishop Button, which lies hard by in the south choir aisle. His sarcophagus, covered by a slab of Purbeck marble, has the figure of the bishop chased upon it, and is the oldest inscribed slab in England.

Bishop Button was the second bishop of Bath and Wells; he died A.D. 1274, and was later canonised. It was at his tomb that the offerings were made which helped to furnish the church, sufferers from toothache and neuralgia coming here in large numbers to be cured by the

saintly influence. Why this bishop should have had the reputation for a power of curing toothache and neuralgia is possibly explained by the fact that he himself, though dying in old age, had a perfect set of thirty-two teeth, with no trace of decay or discoloration.

When his coffin was opened in 1848 his skeleton was found to be untouched, and it was noticed that his teeth were absolutely perfect in every way.

There is, however, reason to believe that caries of the teeth is a disease of comparatively modern development, and though the actual progress of the disease in the dentine

FIG. 28.—Two ancient stone carvings in Wells Cathedral, illustrating facial neuralgia. That on the right is the well-known "Toothache" figure.

of the tooth is no doubt due to a definite organism, it is possible that the frequency of the disease in modern days is due to a change in the habits of the people, as, for example, the enormous increase in consumption of sugar since the days of the Tudors.

Another probable factor is the substitution of soft cooked food for nuts and grain and uncooked vegetables which doubtless formed a large portion of the staple diet of primitive peoples. In Saxon times dental caries was uncommon, and in ancient Egypt the examination of skulls of exhumed mummies shows that caries then was rare. The association between dental caries and paroxysmal trigeminal neuralgia will be discussed presently when dealing with its etiology.

Of the three main divisions of the trigeminal nerve it is

especially the second and third divisions that are liable to the paroxysmal tic. These may be affected in about equal frequency either separately, or both the second and third divisions may be involved at the same time, often one division suffering alone for some years before the neuralgia invades the territory of the other. Only rarely is the first division affected, and then always in association with the second division, though I have known paroxysmal neuralgia limited to the supra-orbital area for nine years before the pain began to invade the upper lip and side of the nose, indicating that the second division was also involved.

Paroxysmal Trigeminal Neuralgia at its onset is difficult if not impossible to distinguish from dental neuralgia, especially when its beginnings are gradual and not violently spasmodic. It is only by the exclusion of dental and other causes, and by the long continuance of the painful attacks and their resistance to ordinary treatment, that the diagnosis of the inveterate neuralgia may become established. In a large number of cases the first onset of the pain is not gradual but *sudden and violent*, the intensity in the first paroxysm being perhaps more severe than in any subsequent attack. Thus, I have known a case of a woman, a cook in domestic service, who was awakened one morning at three o'clock by the most violent and maddening pain in her cheek and jaw, which was so severe that she lost all control over herself and shrieked continually until the whole household was roused, and the doctor had to be sent for; indeed for the next fortnight she had to be kept almost continually under morphine. The pain then left her completely for a while, but returned again and again, and when I first saw her six years afterwards her attacks were most distressing to witness, the paroxysms lasting as long as twenty minutes. She was one of the first patients whom I treated by alcohol injection in 1908 for this disease, and in April 1915 I destroyed her Gasserian ganglion by alcohol injection; her relief, which has already lasted for ten years, is probably permanent. So sudden is the onset of the pain and so violent in character, if one

may judge by the loss of control some patients exhibit, that I have seen a man when sitting quietly in my consulting-room describing his trouble suddenly jump up with a shout, throw himself on the floor, rolling about clawing and tearing at his face and yelping like a maddened animal. After a minute or so of this performance he would suddenly stop, get up and sit down again in the chair and continue talking as before. Such violence and suddenness of the painful paroxysm are characteristic of trigeminal neuralgia, and are scarcely met with in any other condition. Intensity of pain is difficult to gauge, and the same degree of pain that may give rise to an extravagant histrionic demonstration in an individual of unbalanced temperament may be borne with stoicism and with very slight external evidence of suffering by another person. Often it is only those in immediate contact with the patient who realise that the neuralgia is something out of the ordinary, and many sufferers from this terrible complaint get little sympathy from their friends, as ordinary neuralgia is common enough.

After the first onset of the attacks of pain, which may last from a few minutes to a few weeks, there may be *complete cessation for a year or more ;* I have known thirty years elapse between the first and second attacks, in others sixteen years, nine years, seven years, and shorter intervals are frequently met with.

With succeeding attacks the intervals of freedom from pain tend to become shorter, though some patients have more or less periodic bouts of neuralgic seizures for several weeks or months every year. Some may have scarcely any freedom, but from the very commencement have never a day or even a waking hour free from attacks. Such persons have almost continual hypersensitiveness of the face, being afraid to touch or wash it or to brush the hair on that side. One woman indeed that I have treated by injection was for twenty years unable to wash the right side of her face.

The pain is referred to various parts in the upper and

lower jaws, in many cases shooting along the side of the tongue, along the lip and side of the nose and to the back of the cheek or front of the temple. In second division neuralgias in addition to the pain affecting the cheek, upper lip and nose, the neuralgia is often described as affecting the eyebrow and lower half of the forehead, and spreading up to the temple outside the eye. The character of the pain is usually sharp and shooting, and may be likened to red-hot needles or wires, the pain darting along like electricity, and ending like the explosion of a bunch of fireworks in the face. In others, the pain is like knives sticking in, and many will deny any sensation of heat. Different sufferers behave differently during the spasms. With some, the jaw may be fixed wide open, and salivation may be profuse. In others, lachrymation is seen, especially in neuralgia of the first and second divisions. Some press the hand upon the cheek firmly as a mode of arresting the pain, some dare not touch the face, while yet others rub the face hard with the hand, and from the constant friction the eyebrow and moustache may be practically rubbed away, and the skin becomes thickened, and even shows callosities. One man thought he could arrest the pain by rinsing the mouth out with brandy; some whose pain generally starts at the commencement of a meal can control the pain after a while by forcing themselves to continue eating.

Very characteristic of trigeminal neuralgia is the manner by which the attack may be provoked: in some a light breath of air upon the cheek or the slightest touch on the lip or nose will start the paroxysm, though immediately on the cessation of the pain the cheek and nose can probably be rubbed with impunity for a minute or so before it again becomes hypersensitive. Such areas have been termed "*trigger zones*" by Patrick, but, in most, any movement of the face will start the pain, such as eating, laughing, talking, sneezing or blowing the nose. Certain spots were formerly described as "*points de Valleix*," such as over the exit of the infra-orbital nerve

and the mental foramen, which were supposed to be specially sensitive, and touches upon which were said to start the paroxysms of pain. I am unable to confirm this from my experience.

Reflex muscular spasms of the face are naturally the rule during the paroxysms of pain, a facial expression of emotion, but quite apart from these facial movements there is to be seen in a certain number of sufferers from the painful tic definite *clonic facial spasm* limited to the side of the face afflicted with the neuralgia. These clonic contractions of the facial muscles are more marked during the attacks of pain, but are frequently to be seen between the neuralgic attacks. (See "Geniculate Neuralgia," pp. 324–326.)

The majority of patients are comparatively free from spasms at night, no doubt owing to the quiet and absence of movement, the pain reappearing on washing in the morning or at meal times. In others, however, night brings no relief, and the torture may continue day and night for weeks until the severity of the attacks dies away, and an interval of freedom from the neuralgia returns. In some cases the interval of freedom is complete, no sign of neuralgia being present for many weeks or months, but some persons never altogether lose the sensitiveness of the face and slight neuralgia, though the severity of the attacks is in complete abeyance and life is tolerable.

Year after year the disease tends to become more chronic, the intervals between the bouts of pain being shorter, though the intensity and severity of the paroxysms may be less than formerly. Almost invariably the pain lasts until death puts an end to the suffering, and in former years suicide often terminated the story, before modern methods of relief were known. In one instance only have I known the pain to disappear with advancing age, one of my neuralgia patients, a lady of 60, telling me that her father had suffered from the same disease for 25 years until he was nearly 90, and he lived to the age of 101, the last twelve or fourteen years of his life being free from the pain.

Heredity is a curious feature in trigeminal neuralgia major, which I have observed on numerous occasions, fourteen in all, nearly 2 per cent. of my cases, in which the patient and one parent, either father or mother, had suffered from an identical type of neuralgia. Frazier denies the influence of heredity, though he quotes a remarkable instance of a family of twenty-two children under his care whose mother and three of the children were victims of this malady.

In one instance I treated a lady for some years for bilateral trigeminal neuralgia major, when her daughter aged 35 consulted me for pain in the head and lower jaw which had started two days previously as a jumping severe pain near the mental foramen when going to bed. She had noticed that if she kept absolutely still the pain stopped entirely, but any movement of the face started it again, though sleep was not interfered with. The teeth were in perfect condition, and though chronic trigeminal neuralgia seemed probable, especially as eighteen months before she had had a similar attack less severe for about a week, yet I hesitated to inject the nerve until rest and drugs had been tried. This treatment failed to relieve her, and she lost confidence and went elsewhere for treatment, and she was twice injected with alcohol without getting any relief.

Three years later she came to see me again, when the diagnosis of chronic trigeminal neuralgia was absolutely certain. I injected her third division at the foramen ovale, and she was completely relieved of all pain for seven years, until 1923, when the pain recurred and re-injection was performed.

Analysis of several hundred cases seen in private shows a strong *preponderance of females* with true trigeminal neuralgia in a proportion of three females to two males. There is a similar *preponderance of right-sided cases* in the proportion of three against two on the left side. It is difficult to divide the cases into those affecting the second or third division separately, as so many cases affect both the upper and lower jaws on one side, and when the pain

appears to be limited at first to one division the neuralgia may subsequently affect the other.

Only rarely is the first division involved, and then practically never alone, without the pain invading the second division also. It is indeed a fortunate thing that when the first division is involved in the neuralgia the pain is limited to the supra-orbital nerve, and does not affect other branches of the first division, as this is the only branch of the first division that can be successfully treated by alcohol injection.

A man began to suffer at the age of 44 from a stinging sensation on the right side of his forehead whenever he washed or wiped it. After twelve months flashing pain commenced in the right eyebrow whenever he touched that side of the forehead, but neither eating nor talking started the pain at this time. All his teeth on that side were gradually removed without affecting the neuralgia, which continued for three and a half years limited to the right side of the forehead and eyebrow.

For eighteen months he had noticed that the flashing pain seemed to trickle down from the forehead to the side of the nose into the upper lip, which had then become a sensitive or trigger zone, and eating would then start the neuralgia through his tongue touching the right upper lip.

The neuralgic attacks became so severe that he was unable to sleep. He described the pain as being like numbers of knives in the forehead, the pain flickering back to the vertex as a burning sensation. In April 1924 he was sent to me for alcohol injection, and as he was only 49 I decided not to inject the supra-orbital nerve and the superior maxillary nerve, as the relief would probably last only three years or less, and I therefore injected his Gasserian ganglion in order to try for a permanent cure. This necessitated producing anæsthesia of the third division, which had not shared at all in the neuralgic paroxysms, but it is highly probable that, if only neurolysis of the nerve trunks had been done, eventually the pain would have invaded the third division also.

An interesting and most unusual feature of this case was the long period of three and a half years in which the neuralgia was strictly limited to the territory of the first or ophthalmic division of the nerve, the second division being affected apparently only later, and then only as a trigger zone, the pain not being produced by the movements of the jaw in eating.

The average age of onset of two hundred of my cases I found to be just 50 years. The extreme limits that I have met with personally in my practice have been 87 for the oldest and 12 for the youngest. The latter case when I first saw her was a woman of 32, having already suffered for twenty years.

Mr. Barclay, of Newcastle-on-Tyne[1] has recorded the case of a boy with neuralgia major of the fifth nerve at the early age of 10, relieved by intracranial section of the second and third divisions. Other youthful sufferers that I have met with have been three at the age of 16, three at 17, two at 19, and many in the early twenties. The majority commence between 40 and 60.

A woman, *æt.* 34, began to fall about and developed weakness in the right arm and leg, becoming later a typical case of disseminated sclerosis. After two years she had sudden onset of pain in the left side of the face, severe from the first and during the night only, then day and night, of characteristic tic douloureux type. Three years later, in 1916, I injected the left second and third divisions, getting complete anæsthesia of both. In 1920 the left side of the face was still numb and free from pain, when neuralgia suddenly commenced on the opposite side. Two months later, as the attacks on the right side were becoming very severe, I injected the right third division. In December 1925 she wrote, "I have slight neuralgia on both sides of face occasionally, but nothing much; the numbness has gone."

Though the pain in true trigeminal neuralgia never spreads across the middle line, yet the nerves of both sides may be affected, the *disease becoming bi-lateral*. In a few cases the disease becomes bi-lateral soon after its commencement, but in the majority of cases many years elapse before the second side is involved. Usually one side is

[1] *Brit. Journ. of Surg.*, 1922, **9**, 306.

much less severely affected than the other. I have seen a considerable number of bi-lateral cases, thirty-six in all, twenty-seven females and nine males. The type of disease is exactly the same in the bi-lateral cases as in those where the pain is limited to one side, and the same treatment is applicable, with the exception that gasserectomy must not be performed on both sides, owing to the masticatory paralysis and consequent dropping of the lower jaw which would ensue.

Functional Associations.—The greater frequency of trigeminal neuralgia in the female sex has already been referred to.

Although proportional figures vary in different collections of cases, it has almost always been noticed in statistics of this disease in which definite figures are dealt with that a greater number of women are affected, and thus a greater liability of this sex to the disease must be accepted as a fact and not as an accidental coincidence.

This, I think, is to be explained by the greater liability of women to neuralgia generally, and thus although the primary septic focus in dental nerve filaments is presumably equally frequent in the two sexes, in fact very few persons really escape it, yet the other factors concerned in the development of this type of neuralgia, both predisposing and exciting, might determine a much greater frequency of the disease in one sex than in the other.

Although large numbers of cases occur in persons who in no way can be described as neurotic, highly strung or hysterical, yet I have remarked an undue proportion of individuals who have also suffered in the past with migraine, and some who are out of the ordinary temperamentally nervous.

The neuralgia, as already stated, is mostly limited to the anatomical distribution of the fifth nerve or to one of its branches, and thus differs from psychalgia, migraine or other central pains in which the neuralgia may vary in its situation at different times, and may cross the median line, which true trigeminal neuralgia never does (with the exception of bilateral cases which are easily recognised). Thus

although it must at once be conceded that true trigeminal neuralgia is an organic neuralgia, and thus fundamentally different from a migrainous neuralgia, or a psychalgia, yet I am of opinion that the former disease is more easily provoked in an individual with a neurotic heredity than in those of a more stable and equable nervous temperament.

Association with Organic Nervous Disease.—I have met with seventeen cases, seven males and ten females, of typical chronic trigeminal neuralgia major in persons suffering from some form of chronic spastic paraplegia, usually *disseminated sclerosis;* in three of these cases the spastic paralysis present, though of chronic type, was insufficiently distinctive to make the diagnosis of disseminated sclerosis more than probable. One patient suffered from amyotrophic lateral sclerosis, probably syphilitic in origin, and another from primary lateral sclerosis following upon severe concussion. Two other patients combined trigeminal neuralgia with paralysis agitans. One of these was a man of over 60, and the association of paralysis agitans at the degenerative period of life with trigeminal neuralgia cannot perhaps be held to be more than a coincidence.

The cases of disseminated sclerosis, however, in association with trigeminal neuralgia are too numerous to be accounted for by any theory of chance. What the connection between them is I cannot say. On first meeting this combination of paroxysmal neuralgia with chronic cerebro-spinal sclerosis one is tempted to think that the neuralgia must also be of central origin, and that it would not be beneficially treated by any peripheral nerve treatment. In nearly all of my cases the spastic paralysis had been present for several years before the neuralgia commenced, and my experience has shown me that these neuralgias behave just like the others, and are immediately relieved by a successfully placed alcohol injection. One such case of severe paraplegia from disseminated sclerosis in a man of about 35, who could move neither arm nor foot, I saw in the Royal Hospital for Incurables suffering

in addition to his other miseries from severe bi-lateral trigeminal neuralgia of the third divisions. I injected the foramen ovale on both sides at the same sitting, completely relieving his pain, but giving him some weakness in biting for some weeks.

Owing to the bi-lateral anæsthesia of his tongue which was produced by the double injection, his utterance was so interfered with that he could scarcely make himself understood for the first twenty-four hours, though he soon learnt to use his tongue properly, and normal speech returned.

I have never seen an association of trigeminal neuralgia major and locomotor ataxy, which might well be guessed to be a not improbable combination in view of the shooting pains of tabes. The nearest approach to this I saw in 1924 in a gentleman of 55 who had developed an ataxic paraplegia during the last eight years. He had suffered from syphilis many years before, there was bi-lateral complete nerve deafness and the left knee jerk was absent, he could not walk without support, and the pupils were fixed to light. He had been diagnosed as tabes dorsalis by a neurologist, who sent him on to me on account of the development of severe neuralgia of the right third division during the previous twelve months, but he had never had any shooting pains in the limbs or trunk, there was double ankle clonus and typical bi-lateral extensor plantar reflexes. Injection of the right foramen ovale anæsthetised the third division and arrested the neuralgia at once (see p. 394).

Another remarkable case was that of a man who began first to suffer from right-sided neuralgia major at the age of 41, affecting the second division only. After suffering for fourteen years a partial gasserectomy was done, which relieved him for four years. Alcohol injection was then attempted by two surgeons, but no relief from the recurrent tic resulted.

Two years previously, while free of neuralgia, he began to develop symptoms of left cerebello-pontine tumour, with pain over the left eye and vomiting and ataxic gait. When I saw him in 1915 after the recurrence of the neuralgia on

the right side, there was complete anæsthesia of the right third division, but normal sensation over the second division, which was very hypersensitive, indicating the incomplete nature of the gasserectomy. On the left side there was partial tactile anæsthesia and analgesia of both second and third divisions, so that he noticed he was apt to lose his food on that side of his mouth, but he had never had any pain on that side except over the eye. There was left-sided nerve deafness, weakness of the left external rectus and coarse nystagmus, with an extremely ataxic gait and inco-ordination of the left arm, so that it was impossible for him to get about. I injected his right second division at the foramen rotundum, producing total anæsthesia of the whole of this nerve. After two years complete relief the pain gradually recurred and was again arrested by a fresh injection a year later. A strange feature of the case was that during this interval of three years the symptoms of left extra-cerebellar tumour had practically all disappeared, and he had been able to return to business.

Syphilis was apparently excluded by the history and by a negative Wassermann test.

Pathology

The causes of trigeminal neuralgia have been the subject of much speculation and argument. Fothergill ascribed it to cancer, and since his time many observers have asserted that it is the result of chronic degenerative changes in the Gasserian ganglion, such as fibrositis, and arterio-sclerosis. These latter processes are probably a result of senility, and many observations have been recorded of perfectly normal ganglia in this disease. One such I have had the opportunity of examining in a patient who died suddenly of angina pectoris, in 1909, before any treatment for the neuralgia had been done.

At the autopsy, at which I arrived a little late, I found the pathologist cutting up the brain into thin slices in his search for the cause of death, being persuaded an intra-

cerebral lesion had occurred. I counted myself fortunate that I had not performed any injection, as without doubt at that date the death of the patient would have been ascribed to that cause, so little being then known of the effects of injection of the foramen ovale. When at last I could persuade the pathologist to put the brain aside and examine the cardiac muscle the anginal cause of death at once became apparent. The Gasserian ganglion in this case had not been interfered with by any form of operation or injection, and after staining and microscopical section there was no difference to be seen in the ganglia of the two sides, the cells being perfectly normal, though some arteriosclerosis was present, as might have been expected. If arteriosclerosis, however, were the cause of trigeminal neuralgia, how much commoner should the disease be than it is, and yet this neuralgia is met with in hundreds of persons with soft arteries and, as I have already shown, in some persons of comparatively tender age.

In a considerable number of cases I have observed that the neuralgia has first commenced after some *severe dental operation*, difficult extractions and the like, and in many others there has been a history of bad teeth for years. A characteristic example of neuralgia major following directly upon a dental extraction in which a steel instrument was broken and the piece left behind in the upper jaw, I saw in a woman of 70, who had an instrument broken in her right upper jaw twenty-five years previously.

Three weeks later neuralgic pain started in the right upper jaw and continued with slight intermissions as a typical spasmodic neuralgia until I saw her in 1920, and injected the right second and third divisions of the fifth nerve. In 1910, fifteen years after the neuralgia started, she had been X-rayed, and the broken piece of steel was removed from the jaw and an antral abscess drained, without benefiting the neuralgia. On the other hand many cases are met with in which the teeth are fairly good, and the patient has been more than ordinarily free from toothache.

In ten cases I have seen trigeminal neuralgia of the second

division follow *antral abscess*, and comparing these with the cases following dental operations it appears to me highly probable that sepsis is the primary cause of trigeminal neuralgia, a septic neuritis of the dental nerve filaments or other sensory nerve filaments in the jawbone being the neuralgic focus from which the painful stimuli spread. When this septic infection of the dental nerve filaments has taken place extraction of the teeth will not arrest the disease, and indeed it often aggravates the pain, which may spread apparently from tooth to tooth along the jaw as one by one they are sacrificed in the vain effort to cure the patient's miserable condition. These branches of the fifth nerve are the only nerve filaments in the body which are so constantly exposed to septic infection, and these nerves are, almost without exception, the only nerves which suffer from chronic paroxysmal neuralgia.

This theory will explain why it is that the second and third divisions of the fifth nerve, and not its first division, are commonly involved in the neuralgia.

This septic infection may have been acquired from caries of the milk teeth, which may account for neuralgia of this type in an adult whose teeth are far above the average. The preponderance of cases affecting the right side may be explained, as Mr. Warwick James has suggested to me, by the teeth on the left side getting a freer use of the tooth-brush in right-handed people, and thus caries is more frequent on the right side.

The greater proportion of cases in women is probably to be explained by their greater liability to neuralgia as a whole. Cases of bi-lateral trigeminal neuralgia and of neuralgia affecting both the upper and lower jaw of one side are not to be explained by the disease progressing centrally towards the ganglion, but by the special tendency in the individual. Heredity, too, acts in all probability by transmission of a greater liability to neuralgic processes, for it is not all septic nerve filaments that set up paroxysmal neuralgia, or the disease would be commoner than it is.

That the disease must be peripheral in origin, that is to say, that it is a lesion of terminal nerve filaments, is proved by the results of treatment and by the fact that there is never any defect in sensation or any trophic signs of neuritis in a case that has not been operated upon. If the disease were a central neuritis we should certainly expect to find in many cases at least definite disturbance of sensation in some form, either to touch, pin, or temperature. Moreover, if the disease were central, it would be difficult to understand how relief can be obtained by a peripheral neurectomy, or by injection of the nerve trunk on the peripheral side of the lesion.

If, then, septic neuritis is a fundamental basis of paroxysmal trigeminal neuralgia we should expect, in some cases at least, to demonstrate septic organisms, especially streptococci, in the jaw bone. In one such case I was able to prove this point with the help of Mr. Warwick James, who excised a piece of bone from an edentulous right maxilla in a man of 50 suffering from typical tic, and in two cases I have tried treatment by an autogenous vaccine prepared from the bone of the affected side. Up to the present, however, no definite improvement has been obtained from this treatment.

Three exciting causes in particular may awaken a latent potential neuralgia, viz., *violent emotion, chill to the face, and a blow on the jaw or face.*

Emotion may act as the exciting cause. A woman whose child was run over and seriously injured by a motor-car had the news brusquely broken to her, and she set off at once to the hospital to which the child had been taken. Soon after entering the building she was suddenly struck with a paroxysm of pain in her face, quite new to her then, though developing soon after into a typical trigeminal neuralgia.

In 1921 I saw a lady aged 77 who had suffered from neuralgia major of the left third division for twenty-five years, the pain having struck her in the lower jaw on the day after her husband's death, spasms of neuralgia con-

tinuing night and day for more than a month before she got respite.

She had been edentulous for several years before the pain started, and when I saw her twenty-five years later the neuralgia was characteristic of the inveterate type of paroxysmal trigeminal tic. I injected the Gasserian ganglion with alcohol, causing total fifth-nerve anæsthesia and arrest of the pain, which still remained complete and unaltered three months afterwards.

Another lady, aged 78, also edentulous for years, was suddenly struck with violent pain in the left side of the face, in the spring of 1915, shortly after reading of the sinking of the *Lusitania*. The pain was so violent and sudden that she screamed, as she could not understand what was the matter. The pain recurred at intervals until December 1920, when I injected the foramen ovale.

Gowers,[1] when speaking of the repression of emotion, describes the case of a lady whose husband was going to America. She became hysterical and on the point of breaking down into tears, when her sister somewhat roughly forbade her to cry. This had the desired effect of drying up her tears and arresting the outward manifestations of grief, but shortly afterwards she was seized by violent supra-orbital neuralgia, lasting for several weeks. Another case he quotes of a girl of 18 who "was much startled and alarmed by the unexpected discharge of a gun close beside her. The same evening facial neuralgic pains came on, and continued for five years in most violent paroxysms, sometimes on one side, sometimes on the other. In this connection it may be noted that the fifth nerve is especially related to emotion, both by influencing the secretion of tears and also as the sensory nerve of the chief region of emotional display, the face."

Emotional causes may also act in the reverse direction and arrest a pre-existing neuralgia. It is recorded of Sir John (afterwards Lord) Lawrence, who was a sufferer from chronic facial neuralgia, that the pain disappeared

[1] "Diseases of Nervous System," 2nd ed. vol. ii. p. 796.

on his hearing of the outbreak of the Indian Mutiny, though he suffered from it again in later years.

Well known is the effect of *chill and cold wind* in aggravating facial neuralgia, and in more than one instance I have met with typical trigeminal neuralgia whose first onset followed immediately after a severe chill to the face. In one remarkable instance a woman aged 53 was driven by her son in an open dog-cart for some 20 miles across Dartmoor to Exeter one bleak wintry day while a north-east blizzard was blowing fine snow on one side of her face throughout the journey. This drive was indeed most tragic in its results, for the woman, after being thoroughly warmed and having supper with her son, was seized by intense facial pain on the side which had been exposed to the wind and snow throughout the drive.

Her son, too, was taken ill the same night with pain in his chest, and died of rheumatic pericarditis within a fortnight. His mother's neuralgia became chronic, developing into a typical trigeminal neuralgia.

Cold wind and draught are perhaps the greatest enemies of sufferers from chronic trigeminal neuralgia, and I have known patients who never went out of doors except during the summer months. For this reason patients suffering from this disease are sometimes ordered to warm climates, but hopes of escaping their tormentor in this way are usually fallacious, and I have known many cases whose painful paroxysms were just as severe in hot summer weather.

Trauma is a definite factor as an exciting cause of neuralgia major of the fifth nerve, and I have already referred to the large number of cases of the disease which commence after difficult dental extractions.

Presumably the chances are equal for either sex. External violence in the shape of blows upon the jaw, one might expect to be more frequent in the male sex, but I have seen perhaps not more than eight cases in which the neuralgia directly followed a blow upon the jaw or side of the head, too few a number to generalise from as regards

proportional numbers for the sexes. Though external violence is apparently rare as an exciting factor, about 1 per cent. in my cases, yet its association was very striking and unmistakable, as, for example, in the case of a girl who fell off her bicycle, striking her lower jaw upon the ground, a blow which, in her words, dazed her and made her teeth rattle. There was considerable bruising and tenderness of the jaw for a fortnight, when paroxysmal neuralgia of the right third division commenced.

A gentleman, *aet.* 50, when salmon fishing one day in Scotland, was casting with a heavy brass "minnow" when he struck himself a violent blow with it over the left eyebrow, just to the left side of the supra-orbital notch, cutting the skin. He soon recovered from the blow, and thought no more of it until, a few months afterwards, he suddenly felt two intensely severe jabs of pain at the site of the injury, so sharp that he fell forward, thinking for the moment "that a needle had been hammered into his brain." He felt nothing further of the neuralgia for three years, when he experienced similar intermittent pain for ten days. This again passed off for ten months, when it recurred, and he then noticed that the painful spasms left behind a burning sensation in the left side of his nose and cheek, and that eating or talking or handling the face would start the neuralgia. His pain was practically limited to an area on the left side of the forehead over the eyebrow and extending as high as the normal hair line. Even between the paroxysms of sharp pain he was conscious of dull pain in this region. The results of treatment proved the neuralgia to be entirely second division in origin, though it closely resembled in many ways a supra-orbital neuralgia. When the latter, however, is involved in true trigeminal neuralgia the pain shoots up to the vertex, and is not limited to the forehead. Alcohol injection of the supra-orbital nerve at the notch in this gentleman's case produced fairly dense anæsthesia of the forehead and top of the head, without affecting the neuralgia, which spread behind the eyeball. Injection of the second division at

the foramen rotundum instantly relieved all his neuralgic symptoms.

In another even more remarkable case a shop girl aged 23 dropped a coin on the floor of the shop, and a friend helping her to search for it suddenly raised her head sharply and struck the girl upon the lower jaw a violent blow with the back of her head. The force of the blow made her feel as though all her teeth had been shaken loose, but after a few minutes she thought no more of it. An hour and a half later, after closing time, she boarded a bus to go home, going up on to a deck seat. She had scarcely sat down when she was suddenly attacked by the most piercing pain in her face, so violent that she lost all control and shrieked and threw herself about, so that she had to be removed from the bus and put into a taxi and taken home. The paroxysms of pain returned again and again, though rather less severely. When I saw her seven months later the case was clearly one of paroxysmal trigeminal neuralgia; the pain was referred to the third division of the nerve, and was on the same side of the jaw that was struck by her friend's head. I have seen some half-dozen other cases with somewhat similar history of blows on the face within a few weeks of the first onset of the pain. It seems to me unlikely that the blow alone is a sufficient cause for the neuralgia, or that a normal nerve could possibly react in this way to such an injury. If, however, a septic focus is present involving nerve filaments, even though no pain has previously been felt, it is perhaps understandable how a blow may act as the exciting factor. The point, however, which is very difficult of explanation on any theory of causation of the pain is the intense violence and suddenness of the initial paroxysm in many cases, nor have I any theory to offer regarding this.

A curious case was that of a farmer aged 59, whom I saw in 1921, who for forty years had suffered from recurrent paroxysmal neuralgia of the right auriculo-temporal area, following a *lightning stroke* when he was aged 19. He was standing in the middle of a field during a thunder-

storm when he saw a bright flash just in front of him, and instantly felt a blow as with a hammer on his right temple. He was not burnt, and must have got the return shock through his body, not the direct flash.

Ever since that day his right temple has been excessively sensitive to chill or draught, and light touches on the temple, or excitement or noise would start paroxysms of neuralgia, though eating did not affect it. Injection of the foramen ovale gave him very considerable relief, though some pain and sensitiveness remained.

Differential Diagnosis of Chronic Trigeminal Neuralgia

Though in large numbers of cases the recognition of the true form of chronic paroxysmal trigeminal neuralgia is perfectly simple, cases are continually being met with in which this diagnosis has been made in error, the real cause being dental, such as hidden caries, abscess at a root, or an impacted wisdom tooth. Caries is perhaps the commonest and the least excusable source of mistake, though the cavity may be buried beneath the edge of the gum, or hidden at the back of a molar tooth.

Even a dentist's word that the teeth are not the source of the neuralgia should not be accepted in a difficult and doubtful case as an excuse for not personally examining the teeth most carefully, and testing with tapping and syringing them alternately with hot and then cold water.

In one case an X-ray skiagram demonstrated bi-lateral caries, hidden under the gums, in a puzzling case of bilateral neuralgia in a girl in whom ordinary careful inspection of the teeth had failed to disclose any fault.

In another case (see p. 142) related to me by a dentist patient, himself a sufferer from true tic douloureux, he had had under his care when living on the South Coast a gentleman sent down from the North of England because of intractable facial neuralgia for which no cause had been found. Suspecting the teeth, he examined them with mirrors most carefully without result, but on testing each

tooth separately by syringing with hot and then with cold water, he found one molar which reacted sharply with pain. He therefore extracted the tooth, and on examining it he found a minute hole in it, hidden below the level of the gum, penetrating deeply into the tooth and sticking in the hole was the bristle of a toothbrush.

Pain referred to the ear and commencing in the ear and spreading forwards along the side of the cheek and into the lower jaw and neck, even sometimes to the shoulder and down the arm to the fingers, is highly suggestive of a lower molar tooth as the cause. Such cases very rarely give any trigger zone, and the pain is often worse at night, while the paroxysms are less sudden in onset and last longer than in true trigeminal neuralgia. An impacted wisdom tooth is an occasional though rare source of chronic facial neuralgia, though unerupted wisdom teeth are not infrequently found on X-ray examination. In one case of a girl of 20 whom I was asked to see and to inject with alcohol for violent facial neuralgia, the pain was due to cutting the right lower wisdom, and the tough gum had formed a purple swelling over the tooth that was trying to force its way through. For three weeks she had suffered severe and increasing pain, so intense that she had tried to throw herself out of a window. Incision of the swollen gum gave complete relief.

Skiagrams of the jaw in cases of chronic paroxysmal trigeminal neuralgia have frequently disclosed an entire absence of wisdom teeth, though in a case of atypical facial neuralgia an X-ray picture should always be taken. Antral abscess may simulate a paroxysmal neuralgia of the second division, just as frontal sinus distension or suppuration may simulate a primary supra-orbital neuralgia. In both these cases the onset of the pain may be periodic, starting in the later morning hours and lasting until late afternoon. Such long duration of painful paroxysms is never met with in true chronic trigeminal neuralgia. Chronic osteitis of the mandible or maxilla is undoubtedly an occasional cause of intractable facial neuralgia, though, failing improvement

as the result of autogenous or streptococcal vaccine treatment, relief is probably only to be obtained by destroying the nerve trunk as in true trigeminal neuralgia.

In all these types of neuralgia the source of the pain is peripheral, and therefore there will be no trace of anæsthesia upon the skin or mucous membranes, nor any other sign of trophic damage to the fifth nerve or its branches. In new growths or gumma, however, of the naso-pharynx or maxilla, or in tumours damaging the fifth nerve at the base of the skull, or with intra-medullary lesions injuring the sensory tracts of the fifth nerve, anæsthesia of varying degree will be found, a sign which absolutely distinguishes such cases from true trigeminal neuralgia, unless the case has already been treated by neurectomy or alcohol injection.

CHAPTER IX

TREATMENT OF TRIGEMINAL NEURALGIA

After eliminating dental causes, sinusitis, impacted wisdom teeth and other peripheral causes of neuralgia, and having first removed any septic teeth and stumps, the question arises as to whether drug treatment, alcohol injections, or operative treatment should be tried. It is true that occasionally cases of genuine paroxysmal trigeminal neuralgia are met with in which the severity of the paroxysms is only slight, and the patient will refuse anything more drastic than medicinal treatment. In such cases gelsemium or butyl chloral hydrate, or both in combination, may be sufficient to keep the pain in check, but in the majority of cases in which the pain is of the usual fierce type, these remedies fail lamentably to control the neuralgia. Even morphine alone is of no avail, though in emergencies or severe crises of the pain morphine in combination with hyoscine will probably bring relief and sleep. There is a real danger of starting the morphine drug habit if used as a remedy for this disease, an unfortunate combination for an individual suffering with trigeminal neuralgia whose pain the morphine fails entirely to relieve. The usual analgesics, such as aspirin, phenacetin, antipyrin and the like, are of little service, though in combination with medinal or other hypnotics, better nights may be secured. Electricity either in the form of high frequency or ionisation to the face, and sometimes radiant heat, may occasionally mitigate the neuralgia, though in many cases electrical treatment seems to aggravate the disease. In some cases inhalations of chlorylen will give relief. In others, painting 2 per cent. cocaine solution on the gum or tongue is a useful remedy in old people.

Gasserian Operation

In the ordinarily severe and inveterate cases the only certain remedy is neurolysis by alcohol injection, or a Gasserian ganglion operation.

The latter is usually advocated by surgeons who have familiarised themselves with the technique of this somewhat difficult operation, and in skilled hands the mortality is now practically negligible. In the original operation, as practised by Krause and Hartley, the whole Gasserian ganglion was extirpated, the chief difficulty being the removal of the inner or ophthalmic portion of the ganglion, which is adherent to the outer wall of the cavernous sinus. Another source of considerable trouble in this operation is the arrest of hæmorrhage which obscures the ganglion.

Several modifications of this operation have been tried, and Jonathan Hutchinson purposely removes only the outer two-thirds of the ganglion, leaving the ophthalmic portion, so as to avoid any risk of keratitis and loss of vision in that eye. In all partial gasserectomies, however, there is a liability to recurrence of the neuralgia after some years. I have seen it as early as twelve months after one of Sir Victor Horsley's cases, and in others the immunity may last as long as ten years or more.

If, however, the gasserectomy has been complete, and total anæsthesia persists over all three divisions of the nerve, the cure is permanent and neuralgia does not return. Of late years extirpation of the ganglion has given place to a modified operation in which the sensory root is dissected out from the back of the ganglion, and is then avulsed centrally, after dividing the root behind the ganglion. The origin of this method is usually ascribed to suggestions by Spiller of Philadelphia and van Gehuchten of Louvain, but it was first advocated and practised by Horsley[1] in December 1890. His patient, however, died, and he gave up the method for extirpation of the ganglion itself.

In America the sensory root operation has been standard-

[1] *British Med. Journ.*, 1891, Vol. 2, 1249.

ised and brought to perfection by Cushing of Boston and Frazier of Philadelphia. Frazier indeed claims that the motor root can always be saved, so that operation in cases of bi-lateral trigeminal neuralgia is now possible, without causing jaw-drop from bi-lateral paralysis of the masticatory muscles. Cushing, however, is less optimistic on this point. Keratitis is still a danger from sensory root avulsion, though it is less common than after ganglion extirpation.

Care in covering the eye with a watch glass for the first ten days, and irrigation of the eye with antiseptic lotion such as boracic, and the use of 10 per cent. argyrol drops, will in the large majority of cases keep the cornea clear. In some, however, especially those eyes in which conjunctivitis is already present, keratitis ensues in spite of the most careful prophylaxis. When this occurs the eyelids must be sewn together after paring the middle third of their edges, and the eye kept closed for three months or more.

The early recognition of keratitis is important, a steamy or ground-glass appearance of the cornea being one of the first signs, in association with uveal injection around the border of the cornea, and general reddening of the conjunctiva. Loss of the superficial epithelium of the cornea is usually present at this stage, and instillation of atropine followed by closing of the eyelids by firm bandaging or strapping for twenty-four hours will allow complete repair of the superficial corneal damage. If, however, the eye is neglected and the lids are not closed, the superficial ulceration rapidly becomes deeper, and permanent scarring will result, or even perforation of the cornea and total loss of vision in the eye.

Treatment by Alcohol Injection

In 1902 Pitres and Verger[1] and subsequently in 1903 Schloesser of Munich advocated alcohol injection of the deep foramina of exit of the main fifth nerve branches from the skull.

Schloesser employed 80 per cent. alcohol, a drug which in its concentrated form coagulates albumin, and therefore

[1] *Soc. de Méd. et de Chir. de Bordeaux.*

destroys locally any nerve trunk into which it is injected. Alcohol when injected into the tissues causes later the development of fibrositis, and subsequent injections may be hindered by the toughness of the tissues impeding the facility for manipulation of the needle when searching for the nerve. It is therefore a point of some importance to avoid injecting alcohol if possible except into the nerve trunks themselves. The effect of the alcohol is to produce neurolysis, that is to say, a local destruction by coagulation of the nerve fibres themselves at the point of injection, followed by degeneration of the nerve elements below this point to the periphery. So long as the nerve fibre above the point of injection and its nerve cell of origin within the ganglion are intact, a new nerve fibre will at once commence to grow downwards from the proximal undamaged end of the nerve in the process of nerve regeneration, and by this means the damaged nerve is renewed and its conducting powers are re-established after an interval varying from six months to two years.

A complete and thorough injection of the second and third divisions of the fifth nerve causes total anæsthesia to touch, prick, temperature and pressure over most of the territory supplied by these nerves, and in addition injection of the third division at the foramen ovale causes complete loss of taste instantly on the anterior two-thirds of the tongue on that side in the very large majority of cases. Motor paralysis, too, of the muscles of mastication will also result from this destruction of the third division of the fifth nerve, so that the lower jaw will deviate towards the paralysed side on opening the mouth, owing to the weakness of the external pterygoid muscle, and no contraction of the temporal and masseter muscles will be apparent on clenching the teeth. In many cases there is temporary wasting of these muscles, so that there is slight hollowing above and below the zygoma, but very rarely is this permanent, even after a total destruction of the ganglion by injection, though after a Gasserian operation this motor paralysis is nearly always lasting.

For the alcohol injection to be successful the point of the needle must puncture the nerve trunk so that the spirit actually penetrates amongst the nerve fibres. Inasmuch as strong spirit is a tissue fixative, its effects are strictly local, and consequently alcohol will not diffuse into the nerve trunk if injected outside the sheath, unless very excessive quantities are used. In this way alcohol behaves entirely differently from novocain and cocaine solutions, which are commonly used for infiltration anæsthesia. If two per cent. novocain solution is injected into a nerve trunk, dense anæsthesia of the distribution of this nerve will be found after half a minute, but infiltration will take about twenty minutes to produce complete anæsthesia. Alcohol injected into a nerve produces anæsthesia almost instantaneously. Such an injection is, however, excessively painful, the pain resembling a flame or intense burning sensation over the area supplied by the nerve.

Schloesser's original method was to reach the foramen rotundum and foramen ovale from within the mouth, the needle being passed upwards behind the palate through the pharyngeal roof. In 1906 Levy and Baudouin devised the method of approach to the foramen ovale and foramen rotundum through the outside of the cheek, and this route has practically entirely superseded the intra-oral method of approach. They recommended eighty per cent. alcohol mixed with a small percentage of stovaine, but this addition of the analgesic drug can scarcely mitigate the furious pain produced by the injection of the first few drops of alcohol solution. The method I invariably practise is to use a two per cent. solution of novocain, and, when the nerve has been struck by the point of the needle, to inject the nerve with a few drops of this solution, which is a far less painful procedure than injecting a sensitive nerve with alcohol direct. Within half a minute the cutaneous distribution of the nerve will become anæsthetic to a pin point, and the appearance of this anæsthesia is proof that the needle point is rightly placed within the nerve. A fresh syringe is then fitted on to the needle, taking great care not to

displace the needle in any way, and ninety per cent. spirit is then injected, two or three drops at a time, until the area of the nerve is totally anæsthetic to pin, touch and pressure, and until the injection of a few drops no longer causes a burning pain in the cutaneous area supplied by the nerve. I use stainless steel needles, seventy millimetres or two and three quarter inches in length, and one millimetre outside diameter. The point should be short, and the needle is fitted with a mount, with a cone-shaped bore to fit closely on the nozzle of a Record syringe, the shaft of the needle from the tip of the point to the junction of the needle with the mount measuring seventy millimetres. The mount is nickelled and fitted with four transverse milled ridges to afford a

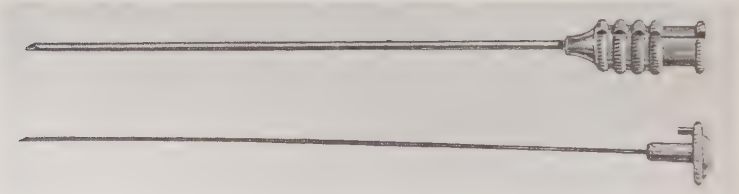

Fig. 29.—2¾ inch steel needle, with stilet.

good surface for holding in the fingers, and these ridges are filed with a groove in the line of the bevelling of the point. A stiff wire stilet bevelled off at the point and fitting the calibre of the needle closely is also fitted with a short milled mount, to which is attached on its inside a short pointer to fit into a notch cut in the mount of the needle. An important detail is that the shaft of the needle must be fitted accurately without protruding into the cavity of the mount, or it will be extremely difficult, if not impossible, to slide the stilet through the mount into the shaft of the needle. I prefer stainless steel needles to those made of iridio-platinum, because the latter do not keep their shape and are inclined to bend during use, whereas a good steel needle will always spring back into a straight line after distortion. The shaft of the needle is made of

drawn steel tubing, and must be of good quality. They are made for me by Messrs. Weiss, of Oxford Street, in four lengths, $2\frac{3}{4}$, $3\frac{1}{2}$, 4 and 5 inches, the two longer sizes being used for sciatic saline injections, and the two shorter sizes are also useful for lumbar puncture.

First or Ophthalmic Division

In the earlier years of the treatment of trigeminal neuralgia it was customary to attempt injection of the branches of the ophthalmic portion of the fifth nerve as they leave the skull through the sphenoidal fissure. This is, however, a difficult if not impossible procedure, and any attempt at injection of these branches at this spot must be mere guesswork, and is almost certain to cause paralysis of the branches of the third nerve, or of the fourth and sixth nerves, with troublesome diplopia. As a matter of fact, the only branch of the first division that is usually found involved in trigeminal neuralgia major is the supra-orbital branch, and this quite rarely. Pains referred over the eyebrow and over the forehead, even up to the hair line, and over the temple up to the side of the head in front of the ear, are in the very large majority of cases due to neuralgia of the second and third divisions respectively, and the pain will disappear when these nerves are successfully injected. In the few cases in which the ophthalmic branch is truly involved in neuralgia major of the fifth nerve, the second division is always affected sooner or later also, and no injection of the supra-orbital should be undertaken until the second division has been successfully treated. In these rare cases, although the neuralgia around the eye and nose and upper lip will disappear with the production of anæsthesia of the second division resulting from injection of the nerve at the foramen rotundum, yet pain of the usual neuralgic shooting character will persist from the eyebrow up to the top of the head. This remaining neuralgia may be almost invariably arrested by injection of the supra-orbital nerve at the notch. This may be done with or without a general anæsthetic. It is a somewhat

painful procedure, but is quickly over and more often than not a general anæsthetic is not used. Two syringes are prepared, one containing two per cent. sterile novocain solution, the other containing ninety per cent. alcohol or spirits of wine.

A hypodermic needle is fitted on to the novocain syringe and the supra-orbital notch is marked by the index finger-nail of the left hand pressed vertically upon it while the hypodermic needle is slipped along the finger-nail down into the notch, just below the eyebrow, at a distance of about an inch from the middle line.

If no general anæsthetic is used, as the point of the needle hits the bone the patient will experience a sharp twinge of pain up to the top of the head. Keeping the needle quite steady, two or three drops of novocain are injected and then the needle is held carefully in position while the sensation of the forehead is tested by a pin point. If the nerve has been correctly injected anæsthesia will be noticeable within half a minute or less. The novocain syringe is then carefully detached from the needle and the alcohol syringe fitted on, and drop by drop about ten minims are injected slowly. In many cases complete anæsthesia of the forehead and top of head results up to the middle line, though the central triangle of skin between the eyebrow and the middle line usually escapes, being supplied by the supra-trochlear branch. In some cases, the anæsthesia is complete even to pressure, while in others only partial anæsthesia results. This is not necessarily the result of an unsuccessful injection, as more than once I have known cases in which a surgeon has cut down afterwards and divided the supra-orbital nerve without increasing the slight degree of anæsthesia already present. This condition is probably due to the fact that the supra-orbital nerve may not be one single trunk, but may reach the forehead as several branches.

In rare cases pain may persist at the vertex over a small area of skin after a successful injection of the supra-orbital nerve, in which all the remaining shooting pain has

been cured. This area of skin is perhaps hardly larger than a shilling or florin in size; it is excessively sensitive to touch and is the site of spontaneous neuralgic pains. I have seen only two such cases, and the causation is a mystery to me, but in both of them the pain disappeared at once and permanently upon giving an additional hypodermic injection of alcohol, pressing the needle down to the bone at two or three spots so as to cover the area affected.

Injection of the supra-orbital nerve produces no anæsthesia along the distribution of the ophthalmic branch to the side of the nose, nor is there any anæsthesia of the eyeball such as results from total ganglion destruction; nor are any trophic results noticeable such as greyness or falling out of the hair which often follow zoster of the ophthalmic division.

In only one case have I seen long-standing paroxysmal neuralgia major affecting the supra-orbital branch and the second division cured of the neuralgia by an alcohol injection of the supra-orbital nerve only. This was in a woman of 54, a sufferer from asthma, who, eight years before I saw her, began to have slight pain over the forehead above the hair line.

Using an asthma cure soon after, she sneezed and immediately after felt "as though something snapped, with a sudden dreadful pain all over the head on both sides; thought she was going to die." At once the pain began to settle down on the left side of the forehead, and continued from that time as a paroxysmal pain like a sharp stab starting at the outer side of the eyebrow, spreading back over the forehead and top of the head, around the eye, and along the nose, cheek and upper lip. The pain would be started by talking or eating, and was aggravated by a cold wind or by handling the face; she said the pain was like boiling water on raw flesh. When the pains were about she could not bear a comb on her hair, the most sensitive spot being the left side of the forehead and eyebrow.

The neuralgia had been getting more severe for the last three or four years, but there was no greyness or loss of hair.

Another sensitive spot or trigger zone, which when touched would start the pain above the eyebrow, was the left side of the chin below the lip. Alcohol injection of the supra-orbital notch cured her pain, but her asthma returned very badly, though it had disappeared during the years of her neuralgia. This may perhaps be looked upon as an example of an alternating neurosis.

In a lady, *æt.* 67, who had suffered for nine months, spasmodic neuralgia persisted in the forehead and eyeball after the second division and supraorbital nerve had both been injected, with resultant dense anæsthesia of the forehead and cheek. The painful spasms did not cease until I completely destroyed the Gasserian ganglion by alcohol injection.

In a recent case, a lady *æt.* 68 had suffered for eleven years from sudden sharp excruciating pains behind the left eye up the forehead to the top of the head, where the pain appeared to explode. Though these attacks were frequent, with never a day's freedom, the neuralgia was limited to the first division of the fifth nerve for nine years before it began to invade the second division. In 1923, as she was unable to wash the left side of her face or to eat without pain, she was sent to a neurologist, who gave her alcohol injection under chloroform. Some improvement lasted for a year, when pain again became severe, and the nerves were re-injected in April 1924. Again there was improvement, though some pain continued until the end of January 1925, when severe neuralgia returned, shooting through the left upper jaw and up to the top of the head. When I saw her in February 1925, the left cheek was intensely sensitive to touch, and there was only a slight trace of anæsthesia remaining on the lower lip and tongue.

She was edentulous, and the frontal sinuses and antra were clear, so I decided the best course would be to inject the Gasserian ganglion. This I did, under local novocain anæsthesia only, pushing the needle through the foramen ovale into the ganglion after I had obtained anæsthesia of the third division to novocain. Not until then did I inject

any alcohol, as my object was to destroy the inner two-thirds of the ganglion connected with the first and second divisions of the fifth nerve, without anæsthetising the tongue and lower jaw permanently. She had never had any pain in the lower division, and the comfort of retaining sensation on the tongue and lower gum and lip would be considerable. This I was able to do according to plan, using 0·5 c.c. of alcohol only, the whole of the upper two divisions remaining totally anæsthetic when seen thirty-two hours afterwards, no sensation even to pressure being present in the forehead, eyeball, cheek, upper lip or jaw, while only slight anæsthesia was present on the third division, pin-prick and scratch being easily felt on the lower lip and tongue. The eye showed no signs of irritation, and the neuralgia had disappeared.

In another case I have seen a true trigeminal neuralgia commence in the first division and remain limited to that division for two years before the pain invaded the second division. In 1905 a merchant captain, aged 31, was working one very hot day in the Red Sea for several hours without sufficient protection to his head, with resultant severe headache on the top of the head and forehead on the right side. Violent neuralgic spasms began six weeks later on the top of his head on the right side, continuing intermittently until, two years afterwards, the pain began to invade the right upper jaw at the end of severe bouts of the frontal headache. About this time also he had appendicitis and developed the heroin habit, taking as much as twenty grains a day. I saw him five years after the pain originally began, and supra-orbital injection did not arrest the pain either in the jaw or on the top of the head, although the injection produced complete supra-orbital anæsthesia. Injection of the second division, however, at the foramen rotundum caused partial anæsthesia of the cheek, nose and palate and the neuralgia ceased. The heroin habit, however, continued, and eighteen months later he committed suicide in China, probably after a recurrence of the neuralgia.

Neuralgia of the Second Division

Second division neuralgia affecting the upper jaw is the commonest of the three varieties to be met with alone, being slightly more numerous in my series than third division neuralgias, though in large numbers of cases both these divisions are affected, sometimes bi-laterally. In many cases the pain may start either in the upper or the lower jaw, and be limited for a time to the second or third division, though after the lapse of some months or years the pain involves both the divisions. Second division neuralgia is often complained of in the region of the malar bone as a stabbing pain at the back of the upper jaw, or it may be referred by the patient to a point between the two jaws at the back, so that it is difficult to decide which of the two nerve trunks is affected. Usually the pain darts upwards outside the eyebrow, and across the lower part of the forehead, into the cheek below the eye and along the side of the nose and upper lip. These latter areas are often hypersensitive, the lightest touch or breath of wind upon the lip or side of the nose being liable to start the paroxysmal pain. The neuralgia often starts quite suddenly. One patient described this as like a crash on the outside of the cheek; she thought she had burst a vein or a nerve had broken. Another felt a crash on the right side of the face as though she had broken her jaw or a tooth, but not painful; then at every meal afterwards she would get sudden flashes of pain like lightning, the neuralgia playing along her cheek like fireworks, and then exploding. Another described the pain as working up above the eye to the top of the head, ending with a crash like a watch-spring smashing, with flashes of pain like fire. In another the attack commenced suddenly at dinner so severely on the right side of his mouth that he thought he had a paralytic stroke. In another the onset was described as though he had "bitten on to a needle." In another the pain was so sudden and severe that she would scream out and struggle and smash things, and she was sure to get a spasm of neuralgia on the right side of her cheek

if she touched cold water with her right hand. It is very unusual to get a trigger zone on areas other than in the fifth nerve itself.

A common form of onset is for the pain to start gradually or suddenly in a particular tooth, the agony becoming so unbearable that in spite of a dentist's assurance that the tooth is perfect the patient insists on having it extracted. Instead of relief being found, the pain either continues in the socket of the extracted tooth or may appear to shift to the next tooth, so that several teeth may be removed in succession, and yet the maddening torture remains. After a crisis of frequent paroxysms for days or even weeks, the neuralgia may suddenly disappear, and an interval of complete freedom may follow, lasting sometimes many years.

A lady of 65 who was suffering very severely from paroxysmal neuralgia was recommended by a friend to drink port, and being willing to try any remedy that might give her relief she drank a whole bottle of port that evening. This made her feel very ill, but the pain immediately disappeared for six years, except for occasional slight twinges.

When the neuralgia recurs it may still be limited to the area previously affected, or it may attack the lower jaw, or both together.

Quite a common mode of onset is for the neuralgia to develop after a difficult dental extraction, especially if the tooth is broken. Most of the foregoing points are common both to the second and third divisions, but a special association that I have noticed in several cases is the development of second division neuralgia major after antral abscess.

In these cases drainage of the antrum, though very necessary, does not cure the neuralgia, which behaves exactly like other cases of true trigeminal neuralgia in being completely relieved by alcohol injection, or by section of the sensory root, indicating the peripheral origin of the nerve irritation. My view is that antral sepsis in these cases is the source of terminal infection of nerve

filaments in the bone, and thus behaves exactly like dental sepsis, which I consider to be the ultimate common source of the large majority of trigeminal neuralgias. In a certain number of cases of second division neuralgia the pain and hypersensitiveness are entirely superficial, at any rate for some years, being referred to the lip and side of the nose. In these cases it is worth while to inject the infra-orbital foramen only, and I have several times thus got complete relief from pain for four or even five years. Usually, however, after one or two relapses it will be necessary to inject the nerve trunk deeply at the foramen rotundum to secure relief.

Similarly, some surgeons divide the infra-orbital nerve at its exit from the foramen, but a better operation is to expose the nerve in the infra-orbital canal and to avulse it from as far back as it can be reached.

This is a very satisfactory operation both for trigeminal neuralgia major limited to the second division, and for some cases of chronic fixed pain referred to the upper jaw and cheek, which will be discussed later.

Injection of the Infra-orbital Nerve

The superior maxillary nerve, after crossing the spheno-maxillary fossa, enters the back of the orbit, passing into the infra-orbital canal and giving off as it does so posterior and middle dental branches to supply the molars and bicuspid teeth, and to supply the bone of the maxilla and the mucous membrane of the antrum with sensory fibres. Shortly before leaving the infra-orbital canal the nerve, now called the infra-orbital nerve, gives off the anterior dental branch which supplies the canine and incisor teeth, and also communicates with the posterior dental nerve.

The infra-orbital nerve then emerges on the cheek through a hole in the maxilla, known as the infra-orbital foramen, beneath the levator labii superioris muscle, situated about a quarter of an inch below the orbital margin. Here the nerve divides into the palpebral, nasal, and labial branches, supplying the lower eyelid with its conjunctiva, the side

of the nose and mucous membrane of the lower portion of the septum, and the skin of the cheek and upper lip, and the mucous membrane of the mouth. With the nerve the infra-orbital artery leaves the foramen and emerges upon the cheek.

To inject the infra-orbital nerve at its exit from the foramen the patient should be leaning back upon a couch or armchair with the operator sitting or standing behind the head of the couch in the same position as for injection of the supra-orbital nerve. The foramen lies about one inch from the middle line, the three foramina,

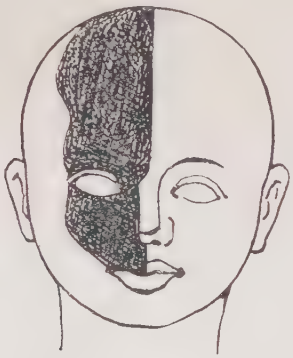

Fig. 30.—Area of cutaneous anæsthesia resulting from alcohol injection of the supra-orbital and infra-orbital nerves.

supra-orbital, infra-orbital and mental, usually being situated on a line almost parallel to the middle line at a distance of about one inch or a little over. A hypodermic needle is fitted on to the novocain syringe and hypodermic injection is made at a point about a quarter of an inch outside and above the angle of the ala nasi, the injection being made down to the bone in an upward and slightly outward direction. The needle is then removed and a somewhat longer and stouter needle chosen, indeed I generally use the same seven-centimetre needle that I use for the deep foramina. This is then pushed in through the hypodermic puncture down to the bone in the same direction upwards and slightly outwards,

but it is important to keep the left forefinger firmly pressed upon the rim of the lower margin of the orbit. This is to ensure that the needle does not slip over the orbital margin and so penetrate the orbit, where an injection would produce considerable œdema, and much other damage might also be done. With the finger pressing upon the orbital margin the needle would at once be felt if it slipped over it. The aim should be to strike the infra-orbital foramen a quarter of an inch below where the finger presses on the infra-orbital margin. The needle can generally be felt to slip into the foramen and it can be pressed along it for perhaps half an inch. If a general anæsthetic is being used the nerve may now at once be injected with alcohol, using about one cubic centimetre. If no general anæsthetic is used, the patient will complain of a twinge in the nose and lip as soon as the needle strikes the nerve. This should be injected with three or four drops of novocain solution, which will be followed within half a minute by anæsthesia of the upper lip and cheek and side of nose. The anæsthesia will end at the inner and outer canthus of the eye, and will extend outwards as far as a slightly curved line joining the outer canthus of the eye to the corner of the mouth. The alcohol syringe should then be fitted on to the needle and about a cubic centimetre injected slowly, a swab being held firmly pressed against the cheek close up to the needle.

This pressure is advisable for two reasons, to control any hæmatoma from the infra-orbital artery, and, secondly, to prevent the leakage backward of the spirit into the levator labii superioris, which muscle has to be pierced by the needle in order to reach the infra-orbital foramen. This safeguard will minimise greatly, if not entirely prevent, any weakness and tendency to drooping of the upper lip. This is the only muscle which is liable to suffer from this injection. If the needle has been pressed back into the infra-orbital canal the injection will also cause numbness of the incisor and canine teeth, and pain may be momentarily felt in them during the injection of the alcohol. The

whole of the cheek is truly anæsthetic as the result of a properly placed injection, though even comparatively light pressure upon the cheek is instantly felt. This is because the jaw bone beneath the cheek is still normally sensitive, but if the finger be passed through the mouth inside the cheek pressure from the outside on the finger will not be perceived, though the alveolar margin and its gum are perfectly sensitive.

As already stated, this injection is sometimes very satisfactory in its results, four or five years complete relief being sometimes obtained. In the majority of cases, however, of neuralgia of the second division infra-orbital injection does not arrest the pain, probably because the origin of the pain is a neuritis of the dental filaments in the posterior part of the jaw. In order to cure this neuralgia, the nerve trunk must then be injected at a point behind the exit of the posterior dental branches, and the best site for this injection will be where the nerve emerges from the skull at the foramen rotundum in the sphenoid bone.

Injection of the Superior Maxillary Nerve

This is the deepest and most difficult of access of the foramina for alcohol injection. Two routes are practicable, and even a third, which may be called the trans-orbital route, has been highly recommended. By this route the needle is passed along the floor of the orbit very near its outer angle, but I consider this method of approach too dangerous.

The two routes which I employ utilise the method of approach to the foramen rotundum through the pterygo-maxillary fissure, and the needle can be passed inwards either in front of or behind the coronoid process of the mandible. Sometimes one, sometimes the other route is the easier. The anterior route is in the more direct line to the foramen, and it is possible for the point of the needle to engage in the lips of the foramen rotundum when passed in this way. Before attempting to pass the needle, guide

lines must be drawn with a skin-writing pencil upon the cheek as landmarks.

First, the anterior border of the coronoid process is felt for, and a mark is made with a pencil at the angle between this and the malar bone above. This is the point at which the needle is to be inserted. The next spot to mark is the upper angle of the malar bone where the frontal process bends sharply upwards. The two points already marked must now be joined by a straight line, and a plane through this line perpendicular to the cheek will pass through the foramen rotundum. Usually the two and three-quarter inch needle already described is amply sufficient to reach the nerve, but for large heads, especially if the cheeks are fat, I use a longer needle, three and a half inches. If it is decided to perform the injection without the use of a general anæsthetic, the skin, after being painted with two per cent. iodine in spirit, is first anæsthetised by puncturing the lower of the two points with a hypodermic needle and two per cent. novocain is injected into the skin and also deeply upwards in the line of the plane referred to. Next, taking the injecting needle in the right hand and holding the skin of the cheek firmly with the left, the point is gently pressed through the cheek and inwards and upwards at an angle of about forty degrees above the horizontal, keeping the line of the needle in the direction of the line drawn on the surface of the cheek. Should the point of the needle hit the coronoid process it must have been inserted a little too far back and this must be adjusted, which can often be done by pulling the skin of the cheek slightly forwards without completely removing the needle. This is then pressed gently but firmly upwards and inwards and the point of the needle will skirt behind the maxilla and will pass through the pterygo-maxillary fissure, which is a vertical cleft about a quarter of an inch in width between the maxilla in front and the anterior edge of the external pterygoid plate of the sphenoid behind. Passing onwards, the needle reaches the superior maxillary nerve at the foramen rotundum at a depth on the average of

from two to two and a quarter inches, though this may often amount to two and a half inches, or even two and three-quarters with a large wide head, especially if the nerve is not found at once and some swelling of the cheek from hæmatoma has taken place. In such cases the longer three and a half inch needle may be necessary, though I very rarely use it.

These two sizes of needles, however, are useful for lumbar

Fig. 31.—Photograph of patient with trigeminal neuralgia of the right second and third divisions. Lines drawn on face to illustrate the point for the insertion of the needle. The lower asterisk in the front line marks the point of entry for the needle to reach the foramen rotundum.

puncture, and the longer needle will be required more often for this operation than for trigeminal injection. Sometimes the needle does not pass straight through the pterygo-maxillary fissure without encountering bone, sometimes the maxilla in front, but more frequently it strikes the external pterygoid plate behind. This is a good landmark for the required depth, as the foramen rotundum is no more than a quarter of an inch deeper, and the needle must then be

gently manipulated forwards until its point is felt to slide over the anterior edge of the pterygoid plate.

Pushing the needle onwards to a depth of another three-quarters of a centimetre, the nerve will be hit if the needle has been inserted at the correct angle. This will be signalled by the patient flinching slightly and at once complaining of a pain referred usually to the side of the nose and upper lip. If the pain is referred to the upper jaw and not to the lip and nose it is probable that the nerve has not been struck, but in any case two or three drops of novocain solution should now be injected. If the point of the needle is up against or within the trunk of the nerve the injection of the novocain nearly always causes a sensation as of " spray " on the cheek, and this is a very useful indication that the needle is more or less correctly placed. After waiting for some fifteen to twenty seconds, the skin of the upper lip should be tested with a pin for anæsthesia. If numbness has appeared and the pin point is distinctly less sharp than on the other side, it may be assumed that the point of the needle is within the nerve trunk, and the alcohol syringe is now carefully fitted on to the needle and a few drops of ninety per cent. spirit are slowly injected.

If the needle has not shifted, the injection of the alcohol into the nerve destroys the nerve instantly, the anæsthesia at once becoming deeper, while the patient complains of a burning sensation on the cheek, lip and nose. This would be excessively painful if it were not for the previous numbing of the nerve by the novocain injection. Slowly drop by drop more spirit is injected until no more pain or burning sensation is produced, and anæsthesia of the lip and cheek and side of the nose as well as of the upper gum and jaw now becomes total to all forms of sensation, including pressure. When this desirable state of affairs is reached the needle is withdrawn and the puncture sealed with a small piece of sterile gauze soaked in collodion. In many cases, however, the needle does not hit the nerve at the expected depth, and the nerve has then to be searched for by gently withdrawing the needle for about three-

quarters of an inch and then slightly altering its angle, which may have been either too high or too low.

More usually I find the needle is apt to be pointed too high at first, but the nerve is there and can be found, given the required patience. The main trouble encountered in this manipulation is liability to hæmatoma from puncture of the internal maxillary vessels, which also pass through the pterygo-maxillary fissure on their way to the spheno-maxillary fossa. Should any hæmatoma appear suddenly, the needle must be quickly withdrawn and firm pressure applied with sterile pads over the cheek for at least five minutes. The hæmatoma is never serious if treated in this manner, and the operation can then be resumed and the injection completed. Two other points of danger must be mentioned. After the needle-point has passed through the pterygo-maxillary fissure, if it is directed too high it will reach the sphenoidal fissure, which is less than a quarter of an inch above the foramen rotundum, and if alcohol is injected in this region, in all probability troublesome diplopia will be produced by injury to branches of the third nerve, or of the fourth or sixth nerve. Sometimes this diplopia is transient, but it may last for three months. In one case, a patient who had been thus injected by another physician two years previously, I found a typical Argyll Robertson pupil on that side, the sequel of a third nerve partial injury at the time of the injection. The other point to be guarded against is an injury to the optic nerve as it emerges from the sphenoid at the foramen opticum, which is a full third of an inch deeper than the foramen rotundum, but in much the same direction. It is therefore a most useful safeguard to feel the external pterygoid plate with the needle-point before sinking it to a depth of much over two inches, as, measuring with the eye the length of needle still outside the cheek, no gross injury can be produced if the needle is not pushed in more than another third of an inch.

Sometimes the route to the foramen rotundum in front of the coronoid process is impracticable on account of great

development of the mandible and maxilla, the coronoid process coming far forward and a large antrum bulging the maxilla backwards. It will then be found impossible to pass a straight needle in front of the coronoid process and the external pterygoid plate. Indeed in the three large anthropoid apes this is the normal condition, but in man, whose biting muscles have for æons been less and less exercised, the maxilla and mandible have shrunk in proportionate size to the bones of the brain case. The alternative route which I use for injection of the superior maxillary nerve is to insert the needle behind the coronoid process, and for this the most suitable point is the same as that required for injection of the third division at the foramen ovale, which will be described presently. Instead, however, of directing the needle slightly backwards it is directed forwards at an angle of from fifteen to thirty degrees, and upwards at an angle of about fifteen degrees.

In any case, the objective is to hit the external pterygoid plate, and then to manipulate the needle forwards until its point is felt to slip in front of the anterior edge of the plate, when the nerve will be found at a slightly greater depth, as already described. If the nerve is not found at the first attempt the needle must be partially withdrawn, and its angle of direction altered slightly upwards or downwards until the nerve is found, when the subsequent process of injection is followed as above described. Probably there is less risk of hæmatoma when using this route.

Although by this posterior route the needle cannot possibly enter the foramen rotundum, it nearly always hits the nerve behind Meckel's ganglion, as evidenced by the dense anæsthesia of the hard palate and the anterior half of the soft palate, which is the same as when the nerve is injected by the anterior route. It is not of much importance, however, to produce anæsthesia of the palate, so long as the nerve is destroyed behind the exit of the posterior dental nerves. No anæsthesia of the temporomalar region is ever produced by the most successful destruction of the superior maxillary nerve, owing to its

area of supply being overlapped by the auriculo-temporal nerve behind and the supra-orbital in front. This area, however, it should be remembered, is a very frequent site of complaint of pain in second division neuralgias.

During the process of injection of the nerve first with novocain, and then with alcohol, a common source of error is apt to arise during the testing of the skin for anæsthesia. The attention of the patient is apt to wander and she may continue to say " Yes " when asked to say if she feels the pin prick, although the skin may be totally anæsthetic.

This confusion is more liable to occur if the patient can see the testing pin being used, and therefore in order to convert a subjective test into a much more reliable objective test I tell the patient, while the eyes are closed, to call out as soon as she feels the pin prick. By this method of comparing one side with the other, it is easy to make certain whether anæsthesia has appeared or not, and with some nervous patients it is especially necessary to make quite certain that they cannot guess when the pin is being used except by feeling it.

Unless this chance of error is remembered, the nerve may be injected with novocain and properly numbed, and the needle then moved and tried in another direction, before it is recognised that the cheek is anæsthetic.

It will then be necessary to postpone completion of the injection for an hour at least, until the anæsthesia has worn off.

It is a good rule never to inject alcohol until anæsthesia has resulted from a preliminary novocain injection, first because it is useless to do so if the novocain has failed to produce anæsthesia, and also because of the fibrosis that is produced by repeated alcoholisation of the tissues, which will render re-injection at a future date much more difficult. This is one of the reasons why I object to alcohol injection under a general anæsthetic, unless it is done by the method to be described later.

Inferior Dental Neuralgia

Occasionally cases are met with in which chronic neuralgia affects one lower jaw only for years, never spreading into the tongue or involving the lip or temple or upper jaw. Neuralgia of this description may be caused by injury to the jaw from a difficult extraction, or may persist as the result of a periostitis. Such cases may sometimes be successfully treated by injection of the inferior dental canal from within the mouth, at the point where the nerve enters the canal. Such injections of the nerve with novocain at this point are now commonly performed by dentists for the purpose of producing temporary anæsthesia of the lower jaw during the filling of teeth or other dental operations, an angular mount being necessary for the attachment of the syringe to the needle. If permanent anæsthesia is desired, as in the case of chronic inferior dental neuralgia, alcohol should be used to follow the novocain injection into the inferior dental canal, and I have known good results produced. This treatment will, however, be of little or no value in true paroxysmal trigeminal neuralgia, in which much more lasting results will be achieved by injection of the nerve root at the foramen ovale.

Lingual Neuralgia

Persistent pain in the tongue may be due to deep fissures on the surface of the organ, or to ulceration at the side of the tongue from friction against the rough edge of a carious tooth. Gumma and new growth are common causes of severe pain in the tongue. Though the pain in these cases varies in severity at different times, yet there are no complete intermissions lasting for days or weeks as are met with commonly in tic douloureux.

In inoperable cases of malignant growth of the lower jaw or tongue, or in cases in which recurrence has taken place after excision of the growth, the severe constant pain may be completely arrested by alcohol injection of the mandibular nerve at the foramen ovale.

Paroxysmal neuralgia limited to the tongue may occur as a form of chronic trigeminal neuralgia of the third division. Usually the pain in the tongue is accompanied by neuralgic spasms affecting the lower jaw and lip, side of head and face, but in a few rare cases the pain is limited to the tongue itself, though in the suddenness and severity of the paroxysms, and in their intermittence with intervals of complete freedom, the neuralgia is charac teristic of the true tic douloureux. This form of neuralgia may be treated with success by section of the lingual nerve, but alcohol injection of the lingual nerve alone should not be attempted. Far more satisfactory results will be obtained by alcohol injection of the whole mandibular trunk at the foramen ovale.

During the bouts of neuralgia of the third division in which the tongue is affected, it may commonly be observed that the tongue is thickly coated with fur on the side of the neuralgia, the other side being more or less free. This is probably due to the patient eating only on the other side of the mouth and instinctively preventing food from touching the neuralgic side. After the neuralgia has been arrested by alcohol injection the tongue rapidly cleans.

Injection of the Mandibular Nerve, or Third Division, at the Foramen Ovale

The foramen ovale lies at a depth from the surface of the skin of four and a half to five centimetres, on the average, though the depth at which the nerve is struck by the needle will, of course, vary with the size and width of the head and the thickness of the tissues of the cheek. The least depth at which I have found the nerve is one and five-eighths of an inch, or four and a quarter centimetres, while in two patients with big heads I have had to penetrate a distance of two and five-eighths of an inch, or six and three-quarter centimetres, a variation therefore of a full inch in depth. It will be readily understood, moreover, that should the cheek swell unduly with hæmatoma the necessary depth from the surface of the skin will be greater.

These measures are taken for the ordinary route that I follow, which Levy and Baudouin originally described, through the sigmoid notch of the mandible. For many years I have not used for this route a longer needle than seven centimetres, or two and three-quarter inches. If, however, a more anterior route to the foramen be employed, in front of the coronoid process as described by Härtel, then a longer needle will be necessary, of nine centimetres or three and a half inches in length.

Before proceeding to insert the needle it is well to draw on the skin certain landmarks, the under surface of the zygoma, and a straight line joining the incisura notch of the ear to the lower border of the ala nasi.

In the average skull this line corresponds to the lower border of the sigmoid notch of the mandible, and these two lines therefore mark the available space for entering the zygomatic fossa. The next point to determine is the vertical plane which, passing at right angles to the side of the cheek, cuts the foramen ovale. This plane passes through the middle of the tubercle on the under surface of the zygoma, but as it is practically impossible to feel this tubercle even in a very thin subject, I prefer to measure one inch forwards from the middle of the external auditory meatus. Marking this point on the lower border of the zygoma, a vertical line is drawn downwards through it in front of the ear, usually crossing at right angles the line marking the sigmoid notch. If a needle is pushed inwards through this point of crossing and very slightly upwards it should reach the foramen ovale at the usual depth of four and a quarter to five centimetres, but as the foramen ovale is inclined slightly forwards, and the spinous process of the sphenoid slopes outwards from the back of the foramen ovale, it is probably a better line of approach to the foramen to insert the needle about a quarter of an inch in advance of the point referred to, and to aim very slightly backwards so as to hit the vertical plane already described at the required depth. I prefer to sink my needle at the lowest point possible to clear the sigmoid notch, in order

to give the greatest chance of the needle being passed through the foramen ovale if required. Sometimes, especially in edentulous persons, it will be found that the needle strikes the side of the mandible, and even slight manipulation of the needle will be insufficient to enable it to clear the notch. It will then be necessary to make the patient open the mouth and rest the gums on a dental prop

Fig. 32.—Photograph of the same patient as in Fig. 31, with the needle inserted through the foramen ovale in the Gasserian ganglion. The dark stain on the cheek is due to iodine solution.

or gag, or else to insert the needle at a slightly higher point. Before inserting the seven-centimetre needle I always inject the skin and the deeper tissues with two per cent. sterile novocain solution. The larger needle is then only felt as a pressure, until it is reaching the vicinity of the sphenoid. Sometimes these tissues are very tender, and it is difficult to manipulate the needle without injecting novocain first in several places, and waiting perhaps ten minutes. There is not much risk of hæmatoma in

using this route, as the internal maxillary vessels are usually guarded by the mandible and rarely rise above the level of the notch. Should, however, sudden hæmatoma develop, the needle must be at once removed and firm pressure applied for five or six minutes, when the operation can be proceeded with.

When reaching the depth of the foramen ovale, the needle may strike the middle meningeal artery on its way to the foramen spinosum, just behind the foramen ovale, but this is of little importance, except that should the needle become full of blood it is always best to withdraw it and to clean it thoroughly before re-inserting the stilet and proceeding, because strong alcohol forms a very firm coagulum of blood at once, choking the needle with firm clot. If no general anæsthetic is used the patient will usually flinch slightly when the needle touches the inferior maxillary nerve, and may at once intimate a twinge felt in the lower lip, chin or tongue. Rarely is this twinge felt as a "spray sensation." Sometimes pain is referred to the ear. If on inquiry this is said to be on the surface rather than deep in the ear it is probably the auriculo-temporal nerve which has been touched, and an injection of novocain will be safe to use and will very likely produce anæsthesia in front of and above the ear, or even of the whole territory of the third division. If, however, the pain is referred to the depths of the ear, it is better to make no injection and to withdraw the needle about one inch before pushing it in a slightly anterior direction. Pain referred to the depth of the ear in these cases usually indicates that the Eustachian tube has been touched, and although an injection of novocain into this tube will probably cause no harm yet it is better avoided, because if it be anæsthetised by novocain the Eustachian tube will be no longer sensitive to further searching with the needle, and should an injection of strong spirit be made into it serious damage will probably be done to the middle ear, and deafness or even facial paralysis may result from the spirit penetrating the middle ear. This is a serious danger to the in-

experienced operator, especially if general anæsthesia be employed.

When this Eustachian pain is produced it usually means that the needle is directed too far backwards, and the necessary alteration must be made by withdrawing the needle slightly and trying further forwards. If the nerve be missed again slight pain may be felt in the throat, and if the injection be then made the patient may feel it at the back of the tongue.

This indicates that the levator palati and the wall of the pharynx have been pierced by the needle. This is of no practical importance, no harm resulting, but it is an indication that the needle has been pushed too far.

Sometimes, if difficulty is experienced in finding the nerve, it is a good plan to direct the needle slightly forwards and a little downwards so as to hit the external pterygoid plate. This will give a fair indication of the depth at which the foramen should be found, and working the needle gently backwards until it slips off the posterior edge of the plate, the point of the needle should then be raised and directed upwards and a very little further backwards, so as to hit the foramen which lies just behind the junction of the pterygoid plate with the body of the sphenoid.

I have enlarged somewhat on the difficulties which may be experienced in finding the foramen ovale, but in many cases the nerve will be struck with the first insertion of the needle at the average depth and direction, and the whole operation may be completed in a comparatively few minutes.

The important points to remember are to work deliberately, and invariably to use the method of trial and test with novocain, and never to inject strong spirit before anæsthesia to novocain has been produced. If this method is strictly adhered to little or no damage will ever be done, and the nerve will very seldom be missed.

In quite a large proportion of cases when the needle has struck the nerve the patient complains of pain only locally and does not refer to the periphery of lip, tongue, or chin.

This I have noticed in about one-third of my cases, and if one was not aware of the fact that local pain only is complained of in quite a large proportion of cases the needle might be withdrawn and searching continued in another direction, and the nerve never found. To obviate this mischance, I always inject two or three drops of novocain whenever local pain is complained of, unless it is referred deeply in the ear, and I then wait about half a minute before testing the lower lip and chin with a pin. If no anæsthesia has then appeared it will be safe to move the needle in a different direction, but I have so often found, as already stated, that when the needle has been inserted to the usual depth in the average direction the complaint of local pain only may indicate puncture of the nerve at the foramen ovale, as will be indicated by anæsthesia following a novocain injection. As soon as anæsthesia to novocain appears the syringe should be changed for one containing ninety per cent. alcohol, which is then injected about two drops at a time. It is rare for the preliminary novocain injection of the third division to cause any " spray " sensation, though, as already described, this is common enough in injection of the second division. As the alcohol is injected burning sensation appears in the lip, cheek and chin, and a wooden feeling spreads along the lower jaw and in front of the ear. The burning sensation passes off in from fifteen to thirty seconds, and more alcohol should then be injected, two or three drops at a time, until the anæsthesia is complete and no more burning sensation is produced. It is quite common to find after the injection of from half to one cubic centimetre of the spirit that the burning pain spreads to the nose and cheek and eye, and anæsthesia appears over the upper two divisions of the nerve, indicating involvement of the Gasserian ganglion in the injection (see later).

A well-placed alcohol injection of the third division at the foramen ovale causes complete anæsthesia, even to ordinary firm pressure, of the lower lip and chin nearly to its lower border, and bounded on the outer side by a

downward-sloping line at an angle of forty-five degrees outwards from the angle of the mouth. In many patients a strip of cutaneous anæsthesia to light touch and pin, though not to pressure, will be found extending upwards from the angle of the mouth towards the temple, an area supplied by the buccal branches. The area of skin on the temple supplied by the auriculo-temporal nerve will also be analgesic to pin-prick and anæsthetic to light touch, though pressure will be usually perceived, and the external auditory meatus will be somewhat numbed and insensitive,

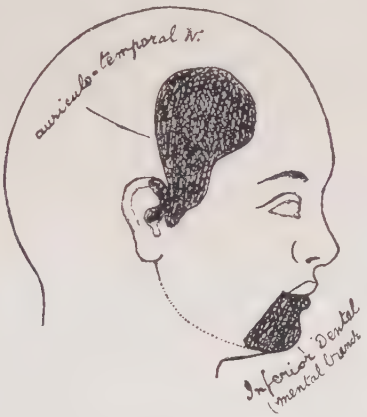

Fig. 33.—Area of cutaneous anæsthesia resulting from alcohol injection of the mandibular nerve at the foramen ovale.

partial anæsthesia being detectable on the anterior wall of the auditory meatus even down to the membrana tympani, which will now be found to have lost its sensitiveness to touch. Inside the mouth the half of the lower jaw and teeth will be numb and wooden, and the gum and inside of the cheek below the level of the teeth will also be anæsthetic.

The front half of the tongue will be insensitive to touch and pin-prick, and in a large proportion of cases taste sensation will be lost completely at first on the anterior two-thirds of the tongue on that side, though a considerable number of cases regain partial or complete gustatory

sensation within a few days. In quite a large number of cases, however, taste sensation is lost on the front half of the tongue so long as the numbness remains complete. Motor paralysis of the masseter and temporal and pterygoid muscles also results from this injection, so that the lower jaw will fall towards the same side when opened widely, though, because the muscles on the other side are unimpaired, eating is not interfered with, as, owing to the anæsthesia produced within the mouth, all eating will be done on the sound side. If the patient is edentulous the false teeth should not be worn for a couple of days as, on account of the numbness of the lower lip and tongue, there is considerable risk of the patient chewing the numbed part between the teeth without feeling it, producing a raw ulcer, though this is not, however, of great importance and always heals well. Practically in every case of third division neuralgia the pain ceases forthwith as soon as the injection is completed, and even though spasms of the neuralgia may be felt while the nerve is being searched for, yet as soon as the preliminary novocain is injected into the nerve the spasms cease. Although the dentures should be removed for a couple of days after successful injection of the third division, yet it is important, if possible, that they should be worn during the injection, as an edentulous jaw in closing raises the sigmoid notch appreciably.

The period of immunity from pain after a well-placed injection is perhaps three years on the average, though I have had numbers of cases of five, seven and nine years' freedom, another of thirteen years and then recurrence of the pain, and in another patient there has been no trouble whatever up to the present time since the third division was injected sixteen years ago. This latter case I saw in June 1909, a lady aged 37 who had suffered from trigeminal neuralgia for over twenty years, the disease having commenced at the age of 17. After three years of pain a surgeon divided the inferior dental nerve, which gave her two years' relief, but the neuralgia then gradually returned and had been severe and persistent for twelve years until

I saw her in 1909. One injection only was given to the foramen ovale of eighty per cent. alcohol, no eucain or novocain being used. The nerve was well struck and complete anæsthesia of the third division was produced, traces of which were still present in 1924.

In my experience injections of the third division give an average longer immunity than injections of the second division at the foramen rotundum.

Injections of the second division only rarely cure the neuralgia for more than four or five years, while one to two years is more usual. Total third division anæsthesia resulting from alcohol injection of the foramen ovale never recovers completely, some traces of anæsthesia persisting even in cases in which the neuralgia returns after some years. On the other hand, anæsthesia of the second division resulting from injection at the foramen rotundum, even though total and complete at first, usually disappears entirely after the lapse of two years. The probable reason for this expectation of longer immunity for injections of the third division, is that a well-placed injection at the foramen ovale is likely in the majority of cases to infiltrate the ganglion partially, so that even though no anæsthesia of the first or second divisions be detected as a result, yet the nerve cells of the outer portion of the ganglion connected with the third division are partially destroyed by the alcohol. I have never seen any trophic damage result from injection either of the second or third division, with the exception of temporary ulceration of the inside of the lip or edge of the tongue produced by the patient unconsciously chewing the anæsthetic part.

Injection of the Gasserian Ganglion

In 1908, when I first practised injections of the foramen ovale on the dead body, by the route I have already described through the sigmoid notch, using one cubic centimetre of methylene-blue solution, and then examining the Gasserian ganglion and the intra-cranial cavity generally in order to trace the course of the injection, I was some-

what surprised to find at first that in nearly every case the Gasserian ganglion was stained blue throughout, and if I worked the needle through the foramen, not only was the ganglion stained blue, but also the wall of the cavernous sinus, often on both sides, and even the ventral surface of the pons.

It is therefore not difficult to understand that rapid injection of one cubic centimetre or more of alcohol within the foramen ovale may cause considerable cranial nerve paralysis. Indeed, a case has been put on record in which an injection through the foramen ovale by Schloesser's intra-pharyngeal method produced almost immediate paralysis of all the cranial nerves on both sides except the second pair, and left as a permanent result considerable cranial nerve paralysis, including almost total bi-lateral deafness, and both trigeminal nerves.

In another case of a girl aged 20, which has been published both in America and in this country, a physician attempted injection of the ganglion under an anæsthetic for chronic neuralgia of the second division: partial destruction of the ganglion only resulted, the inner portion being totally anæsthetic, leading to ulceration of the cornea and loss of vision, but the alcohol must have penetrated the lateral recess and reached other cranial nerves, so that sixth, seventh, and eighth nerve paralysis on the same side was produced, which remained permanent. Unfortunately, too, the neuralgia returned after six weeks, and a Gasserian operation was later performed, and the sensory root of the fifth nerve avulsed, finally curing the neuralgia. Ganglion injection is therefore an operation not to be attempted without great caution and much experience, and distressing results such as the case I have just quoted almost justify Cushing's [1] strictures upon injection of the ganglion.

In my first publications [2] on the subject of alcohol injections I referred to the possibility of injecting the Gasserian ganglion, an operation which I first performed

[1] *J. Am. Med. Assoc.*, 1920, 75, 441.

[2] *Lancet*, May, 1909, 1, 1310, and *Proc. Roy. Soc. Med.*, Feb. 25th, 1909.

for a patient with trigeminal neuralgia in November 1910, using the same route through the sigmoid notch as for injection of the third division. In 1909 I wrote: "If the needle is pushed in too horizontally the wall of the pharynx will be pierced at a depth of from two to two and a quarter inches, or again, if it be pushed too far through the foramen ovale, the cavernous sinus and internal carotid artery will be punctured. To avoid this the needle should never be passed through the foramen to a greater depth than two and a quarter inches from the surface, even in *those cases where it is thought advisable to inject the ganglion itself.*

"If the neuralgia is limited to the third division of the nerve it will be sufficient usually to inject the nerve itself at the level of the foramen, without passing the needle through into the ganglion."

I also showed that injection of the foramen ovale in the cadaver, using one cubic centimetre of methylene-blue solution, almost invariably stained the whole of the ganglion deep blue, and not the ganglion only but also sometimes the wall of the cavernous sinus, the anterior surface of the pons and even the opposite cavernous sinus to a less extent. Such wide spreading of the alcohol explains how cranial nerve paralyses may occur when the injection is done too rapidly, and how headache at the base of the skull and pain on the opposite side of the head may be produced during the injection. When such symptoms are complained of it indicates leakage of the alcohol beyond the ganglion, and no more should then be injected.

In January 1912 I published[1] the results of seven cases of alcohol injection of the Gasserian ganglion, giving details of my technique. Four months later, in May 1912, Härtel,[2] from Bier's clinic, published his method of anæsthetising the Gasserian ganglion by novocain infiltration, for the purpose of rendering facial operations painless without a general anæsthetic, quoting my paper in the *Lancet* of 1909, and two years later, in 1914, he published his

[1] *Lancet*, 1912, **1**, p. 218.
[2] *Arch. f. Klin. Chir. Berl.*, 1912, 193–292.

method as applied to treating trigeminal neuralgia by alcohol injection of the ganglion.[1] On p. 433 in this article he quotes me as having been the first to inject the Gasserian ganglion.

Härtel preferred the longer route to the foramen ovale by inserting his needle in front of the coronoid process, by which means a steeper line of approach to the foramen ovale is obtained and greater certainty of passing the needle through the foramen is assured, once the foramen is reached. It is, however, a much more difficult method, owing to the considerably greater depth from the surface, there is more danger of passing the needle too far through the foramen if it should be found, and there is also an added risk of hæmatoma from injury of the internal maxillary vessels.

The method of approach I use to reach the ganglion is practically the same as for injection of the third division, except that I insert the needle rather below the point referred to on the skin, and if necessary I use a dental prop to keep the mouth open and thus to depress the sigmoid notch.

When the third division has been found by the needle at the foramen ovale and anæsthesia of its distribution has resulted from the novocain, I inject it with ninety per cent. alcohol without moving the needle any further until the third division is completely anæsthetic.

In some cases if a further quantity of alcohol is injected, a few drops at a time, the patient will complain of a burning pain spreading up over the cheek and nose even up to the eye and forehead, which indicates that the alcohol is infiltrating through the root of the third division into the Gasserian ganglion. At this stage the patient often complains of a sensation as of a hair or similar foreign body in the eye, and blinking of the eyelids will be noticed in the spontaneous effort to get rid of the irritation. This is due to the developing anæsthesia of the cornea and conjunctiva, and is a sure sign of the injection reaching the inner portion of the ganglion. In a few cases the whole

[1] *Deut. Zeit. f. Chir. Leipzig*, 1914, CXXVI, 429–552.

ganglion may become totally and permanently anæsthetic with less than one cubic centimetre of alcohol, and without making any further effort to penetrate the foramen ovale. In a larger proportion of cases, with the same indications of the alcohol infiltrating the Gasserian ganglion, it will be found on testing that the first division, forehead and eyeball, becomes totally anæsthetic before the cheek and nose become numbed. Indeed, this happens so frequently as to excite surprise that the inner border of the ganglion should become anæsthetic before the middle portion, when the injection is pushed in from the outer side. Possibly the explanation is that the alcohol runs round the inside of the sheath of the ganglion from the outer side to the inner side before it is able to penetrate into the substance of the ganglion. In quite a number of cases if no more alcohol is injected the anæsthesia will disappear entirely on the forehead and eyeball in from five to ten minutes, a proof that the anæsthesia was not due to a destruction of the nerve cells of the inner portion of the ganglion, but that it was a "shock anæsthesia."

Nerve cells once destroyed never recover. Often the whole area of distribution of the fifth nerve becomes anæsthetic in this way, though within ten minutes the numbness may have disappeared from the first and second divisions, leaving the third division totally anæsthetic. The third division does not behave like the first and second, because the nerve-fibres have been destroyed by the alcohol, in addition to any ganglion infiltration causing this primary shock anæsthesia. If now a few drops more alcohol be injected the anæsthesia may again return over the first and second divisions, which again may disappear three or four times with repeated injections of a few drops, until the anæsthesia remains permanent.

In many cases the first division will remain permanently anæsthetic, while the second division recovers a considerable amount of sensation.

This peculiarity of tendency of the first division to become anæsthetic before the second, during ganglion injection, is

an unfortunate difficulty, because in the very large majority of neuralgias it is the second and third divisions alone that are involved, and if either division is spared it is the first division that we prefer to leave sensitive, in order to avoid keratitis and the risk of visual defect.

I have found in many cases that this can be obviated by pushing the needle rather deeper in through the foramen ovale, perhaps another quarter of an inch, and I have then found that the injection of a few drops of alcohol will anæsthetise the second division before the first is affected, and thus sufficient destruction of the middle portion of the ganglion, with consequent anæsthesia of the second division, can be produced without involving the first division more than partially. So long as sensation to pressure persists on the forehead and eyeball, I find there is no danger of any keratitis. Sometimes some difficulty may be encountered in manipulating the needle through the lips of the foramen ovale, though if a sufficiently steep line of approach be chosen, it is only in rare cases that it will be found impossible to pass the needle through.

Sometimes it will be found after working the needle through the foramen ovale that cerebro-spinal fluid will drip slowly through the needle. This indicates that the point has penetrated through the nerve substance into the cave of Meckel, and any injection made now will not be into the nerve tissue, but into the cerebro-spinal fluid at the base of the brain, and alcohol especially must not be injected at this stage, as it is liable to enter the lateral recess, and to reach other cranial nerves, especially the third, sixth, seventh and eighth. Indeed, when injecting the ganglion fully, after half to one cubic centimetre has been used there is great probability of leakage of the alcohol through the ganglion either into the lateral recess, or along the sensory root into the posterior fossa, and a syndrome I have seen on many occasions is produced in this way. It will be noticed that, while the face is becoming numbed and totally anæsthetic, the patient begins to sway and complains of vertigo; coarse nystagmus is seen and the

face becomes paralysed on the side injected, and in a certain number of cases nerve deafness also is found, while vomiting too is common.

If the injection has been made, as it should be, no more than two or three drops at a time, and if the needle be withdrawn as soon as these symptoms appear, no permanent harm will be done, and the vertigo, nystagmus, facial paresis and deafness usually pass off within three or four minutes, though I have known the nerve deafness last for an hour. In only one of my cases has partial facial paresis remained permanent.

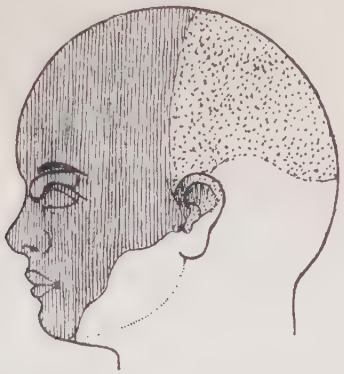

FIG. 34.—Area of cutaneous anæsthesia resulting from alcohol injection of the Gasserian ganglion. The hatching indicates total anæsthesia to all forms, including pressure, the dotted area being partially insensitive to pin-prick and to light touch. The extent of the analgesia to pin-prick behind the vertex as far as the occipital protuberance is unusual.

A certain amount of general headache is often produced during the ganglion injection. More commonly it is referred to the back of the head, and occasionally even to the other side. A dose of aspirin and effervescing caffeine citrate usually relieves this, but the patient must be kept in bed until next day. The diplopia produced in a few cases by involvement of the sixth nerve, or sometimes the third nerve, is usually transient, lasting from a few minutes to a few hours, but occasionally it lasts as long as three months, the longest in my experience being four months, though permanent strabismus has been recorded by other operators.

A possible danger that must be referred to is the risk of

passing the needle too far inwards through the foramen ovale.

I make it a practice never to pass it further than a quarter of an inch after touching the lips of the foramen ovale, but I have been informed by an operator of a fatality at a hospital which he produced by puncturing the cavernous sinus or the carotid artery with his needle. This, no doubt, would happen if the needle were passed too far through the ganglion, but with ordinary care it is not an accident that need be reckoned with.

It is common to find the initial total anæsthesia of the whole fifth nerve distribution considerably diminished in intensity over the first and second divisions within an hour or two, but if the anæsthesia is still total after twenty-four hours it will remain so permanently, except for slight diminution at its periphery.

Injection of the ganglion is probably indicated in those cases in which the neuralgia involves both the second and third divisions, or in those rarer cases in which the first division is also affected. I also think that it is advisable in young subjects suffering from this inveterate malady, as a choice appears to lie between avulsion of the sensory root and injection of the ganglion, for I do not consider that subjects of this disease under the age of 50 ought to be exposed to the necessity of recurring injections every few years or less, but that a permanent cure should be produced if possible. In scores of cases I have found such a result possible to obtain by ganglion injection, and I have many cases now on record who have had no recurrence since the injection of the ganglion ten to fifteen years ago. In a large number of cases of trigeminal neuralgia the pain is limited to the second division, or perhaps the first division with the second, none of the pain ever invading the third division, lower jaw and tongue. When both first and second divisions are involved, the results of injection of the supra-orbital notch and of the foramen rotundum as a rule do not last so long as when the second division alone is involved, the neuralgia perhaps recurring within a year.

Ganglion injection will provide a very satisfactory and often permanent cure for such cases, yet it has always seemed to me a pity to anæsthetise the lower jaw and tongue for these people who never had any pain in the third division.

In 1924 I introduced and practised a modification of the ganglion injection in order to avoid this numbness of the tongue and lower jaw, and have found it very satisfactory. My method is to find the foramen ovale in the usual way, and anæsthetise the third division with novocain so as to prove the position of the needle, and then without yet using any alcohol I push my needle through the foramen for another quarter of an inch into the ganglion, and then slowly, drop by drop, inject ninety per cent. alcohol. This is best done without any further preliminary novocain, so as to avoid unnecessary swelling of the ganglion, and gradually total trigeminal anæsthesia results, perhaps after only five drops of alcohol have been used. The needle is then left in situ with the stilet inserted, and a pause is made for five or even ten minutes, the patient keeping quite still. Usually it will then be found that sensation is returning over the forehead and cheek, owing to the shock anæsthesia of the ganglion cells passing off. A few more drops are again injected, anæsthesia again appearing, and again a pause is made as before.

It may be necessary to repeat this process four or five times before the anæsthesia remains sufficiently deep and permanent, and the whole operation may thus last an hour. The more care that is expended the better and more lasting will be the results. Eventually the novocain anæsthesia passes off from the third division, leaving the permanent anæsthesia of the upper two divisions which has resulted from the alcohol infiltration of the inner two-thirds of the ganglion, and thus the neuralgia will be cured with a minimum of facial anæsthesia, without the uncomfortable numbness of the lower jaw and tongue, and without any motor paralysis of chewing. This latter point is of some value, because if motor paralysis is present, the alignment of any false teeth is liable to be affected, so that the

patient may find it necessary to discard the dentures for a time.

Another complication that is sometimes met with when ganglion injection seems otherwise indicated is that one eye may be nearly blind, the only good eye being that on the side of the neuralgia. Total ganglion anæsthesia from injection is then too great a risk to the sight on account of the possibility of keratitis occurring. In such cases I do not push the ganglion anæsthesia to the point of total loss, for I have found that if sufficient sensibility to pressure remains for the patient to perceive a blunt point on the eyeball and forehead, then dangerous keratitis never develops, nothing worse than slight conjunctivitis ever requiring treatment. I have had such a case to deal with in a woman who had lost her left eye, her neuralgia being limited to the first and second divisions of the right fifth nerve. I therefore performed a partial injection of the ganglion, excluding the third division as described above, with resultant loss to light touch and analgesia over the first and second divisions only. The neuralgia was completely arrested, and the eye was unaffected. It is quite possible, even probable, that such partial ganglion injections will not give permanent relief from the neuralgia, which may recur within a few years, but in that case it should not be difficult to repeat the injection in the same manner, as the absence of third division anæsthesia will offer no obstacle to finding the nerve at the foramen ovale.

There is less risk of permanent paralysis of the motor root by alcohol injection of the ganglion than by the Gasserian operation, as although motor paralysis is produced at first, the muscles commence to recover, with rare exceptions, within a few months, and complete power returns, although the sensory paralysis remains total. Hence in cases of bi-lateral trigeminal neuralgia, of which I have had thirty-six examples, twenty-seven females and nine males, in my series of cases, it is possible to inject the ganglion on both sides with safety as regards the masticatory power, whereas bi-lateral gasserectomy is impossible

on account of the permanent jaw-drop that would ensue. Some of the modern exponents, however, of the sensory root avulsion operation claim that it is possible now to preserve the motor root.

Even in the modern operation of section of the sensory root behind the ganglion, no guarantee can probably yet be given that the motor root will escape, and in fact it is generally divided together with the sensory root.

The reason for recovery of the motor root after injection of the ganglion is that although the motor fibres pass underneath the sensory ganglion and are included in the same sheath, they derive their trophic supply from the motor nucleus in the pons, and will therefore recover after local damage by neurolysis, though the ganglion sensory cells once destroyed never regenerate. Gasserian operations usually destroy the motor root too widely for recovery to be possible. In this way I have been able to perform bilateral Gasserian ganglion destruction by alcohol injection in several cases of bi-lateral chronic trigeminal neuralgia.

In a woman who had suffered severely from right-sided neuralgia for several years, injection of the right Gasserian ganglion in February 1913 caused permanent anæsthesia over the whole trigeminal area, and motor paralysis of the masticatory muscles on that side for some months which subsequently completely recovered. Seven years later she returned to me for neuralgia of the left side and I then similarly destroyed her left Gasserian ganglion so that she is now able to feel nothing whatever on the front half of the face and head. Neither cornea in her case ever showed any sign of keratitis. The masticatory muscles were paralysed at first on the left side, as they had been on the right side formerly, and all these muscles have since recovered and she is able to eat and swallow perfectly. Food, however, she can perceive only by her muscle sense, until it reaches the posterior portion of her palate and fauces, when she is able to taste her food and swallow without discomfort. She has no sense of taste whatever over the front portion of her tongue, all food, as she says,

seeming like chopped straw in her mouth until she begins to swallow it. She is, however, she says, perfectly happy in this condition so long as she never gets the pain again, though she can neither taste her food while she eats it nor, as she says, feel herself kiss her husband.

The cornea must be watched carefully for the first fortnight. Many eyes give no trouble whatever, even though no precautions be taken; this is especially the case with clean brilliant eyes with no conjunctival discharge, but if conjunctivitis be already present before the operation there is then a greater liability to keratitis occurring; in fact, it may be certainly looked for unless the fullest precautions are taken. If not more than 0·5 cubic centimetre of alcohol has been injected into the ganglion itself, and if no conjunctivitis is present, it is probable that no disturbance will result and the conjunctiva will remain perfectly clear. My routine procedure in such cases is to cover the eye with a shade only, which is to be worn both indoors and out for the first week, and outdoors for another week, no bathing of the eye or other treatment being permitted, and most of my cases do perfectly well, without any sign of irritation or inflammation.

One method of protection of the eye that is advised is to cover it by means of a Buller shield or watch glass carefully fastened over it with strapping so as to exclude all dust and draught. A cold wind is undoubtedly a risk to an anæsthetic cornea in the first few weeks, and I have known keratitis set up at once after such exposure in an eye which had remained quite normal for the first week after injection of the ganglion.

It is important not to place a loose pad over the eye, perhaps kept in position by a shade, as the eyelids are then insufficiently fixed and are liable to open underneath the pad, when the anæsthetic cornea may be badly rubbed by the gauze pad without the patient being aware of it. Indeed, more damage can be done by this treatment than by leaving the eye totally unprotected.

In some cases I have noticed while injecting the ganglion

that uveal reddening appears at once, the pupil becomes somewhat contracted and the whole conjunctiva slightly flushed, with a deeper tinge of red around the margin of the cornea. In these cases particular care must be taken to guard the cornea. Atropine should at once be instilled, and the lids kept carefully closed with moist pads covered by oiled silk pressed over the upper eyelid and kept in position by a bandage, while the eye must be carefully bathed twice daily with boracic lotion. If on the second day the signs of conjunctival irritation are not diminished, two drops of ten per cent. argyrol solution should be instilled after the boracic irrigation, as well as a drop of one-half per cent. atropine. This treatment must be continued until all signs of conjunctival irritation have passed, when the bandage and pads may be removed, and a shade substituted. Very occasionally loss of the superficial epithelium of the centre of the cornea takes place within the first three or four days, but when the lids are kept closed and the eye is treated as just described, this shallow ulceration disappears in the course of two or three days. If treatment has been delayed and no precautions have been observed, keratitis may develop and reach the stage of deeper ulceration, which will not heal without some scarring resulting. With this degree of keratitis, it will not be sufficient to keep the lids closed with pads and bandage, but it will be necessary to sew the middle third of the lids together, after paring their edges, and to keep them closed for perhaps six months while the cornea heals.

Recurrence after Gasserectomy.—When the Gasserian ganglion has been totally extirpated, or its sensory root avulsed in cases of true neuralgia major, it is practically certain that the pain will never return on that side, and the anæsthesia resulting from the operation is also permanent in its main features, though, as Morriston Davies[1] has shown, the area of anæsthesia may shrink somewhat within the first few weeks, so that the line of total anæsthesia may finally pass considerably in front of the ear, though at first

[1] *Brain*, 1907, Vol. 30, 219.

the anterior portion of the pinna, and perhaps the concha, may have been included in the anæsthetic zone.

In those cases in which I have met with recurrence of the neuralgia after a Gasserian ganglion operation there has always been considerable return of sensation on the face, especially over the first division, and I have treated many such cases by alcohol injection with complete success.

Jonathan Hutchinson has advocated a partial gasserectomy operation, leaving the ophthalmic portion behind, as he suggests that the pain is never limited to this division, and he states in his book that he has never seen return of the neuralgia after this partial operation. This is too optimistic a view of the partial operation, for I have several times seen recurrence of the pain after such partial gasserectomies by himself and other surgeons. In my opinion, if the patient decides on having a Gasserian operation rather than an alcohol injection, he had much better have the complete operation of avulsion of the sensory root of the fifth nerve, and run the extra risk of keratitis and possibly loss of the eye, rather than be exposed to the danger of a recurrence of the neuralgia after a partial gasserectomy.

After Effects of Ganglion Injection

As already stated, anæsthesia that is still total twenty-four hours after the injection is unlikely to alter much, and in numbers of cases it remains total permanently, just as in cases of gasserectomy, though the posterior margin of anæsthesia may recede forwards from the ear.

Though at first a sensation as if the face were swollen is common, this rapidly diminishes, and usually disappears completely, the patient being conscious of no abnormal feeling whatever. Paralysis of the muscles of mastication is almost certain to be present for some weeks, though this usually recovers gradually after three to six months' time.

The only other trophic effect following Gasserian injection, besides the liability to keratitis, which is treated separately, is the occasional appearance of *herpetic vesicles*

on the lips and chin. With this, some swelling of the lips will be noticed, though no particular discomfort is produced, the part being already anæsthetic. It is doubtless caused by an irritant effect of the alcohol within the ganglion, which is temporary, and is not followed by the post-herpetic irritability and pain that are occasional sequels of zoster in later life.

The prognosis as regards neuralgia after alcohol injection of the Gasserian ganglion is much the same as after gasserectomy. When the anæsthesia remains total over all three divisions the relief from the neuralgia will be permanent, while in those cases in which there is some recovery of sensation, owing to incomplete destruction of the ganglion, there will be a liability to recurrence of the neuralgia after a varying number of years. Cushing and others have pointed to the risks of alcohol injection of the ganglion. In inexpert hands I admit the truth of their contention, yet, as my results show, careful method and deliberation in execution of the injection practically abolish these risks altogether.

On the other side of the picture, gasserectomy in the hands of a surgeon without special experience of this operation may be very dangerous, and even in the most skilled hands I have seen such serious sequels of gasserectomy as hemiplegia, aphasia, and complete change of character, the result of the prolonged compression to which the brain must be exposed in the process of locating and clearly defining the Gasserian ganglion or its sensory root.

Employment of a General Anæsthetic

On the Continent and in America general anæsthesia is practically never employed for alcohol injection, and in theory its use would render the injection a haphazard procedure, as no signal could be given by the patient when the nerve was struck. I have, however, been accustomed, since I first used this treatment in 1908, to employ light chloroform-ether anæsthesia if the patient desires it.

Many patients are so racked with pain, and their nervous control is so reduced by long-continued suffering that it is much better to employ a light general anæsthetic than to expose them to the nervous dread they may have of bearing the additional pain of the operation. People vary so much individually; the majority of cases I do with local novocain anæsthesia only, and such pain as there may be is quite easily borne, less severe than that experienced in the hands of a dentist in the filling of a tooth, but another may feel intolerable tenderness whilst the nerve is being found, and light general anæsthesia is then advisable. If the following technique be adhered to, results as good as those obtained without a general anæsthetic may be had, while the injection is carried out more rapidly, and the patient feels little or nothing of the operation.

Nitrous oxide gas, or gas and oxygen are not very suitable for alcohol injection of the deep foramina, owing to the interference of the face-piece with the manipulations of the needle. I prefer mixed chloroform and ether given on a light mask, the anæsthesia being just deep enough to prevent the patient feeling the insertion and manipulation of the needle, but not deep enough to abolish the conjunctival reflex entirely. When the needle has reached the approximate depth, even less anæsthetic can be given, and when the needle strikes the nerve a characteristic flinch of the facial muscles is seen, though the patient knows nothing of it afterwards. The anæsthetic is then at once stopped, and an injection of a few drops of novocain two per cent. solution given. With a well-managed light chloroform-ether anæsthesia the patient will quickly recover consciousness sufficiently for testing purposes, and while the needle is carefully kept *in situ*, pin-pricks are then administered to the sound side of the face, and as soon as the patient flinches a similar pin-prick is applied to the treated side. If no sign of reaction is produced on repeated testing, though on the other side of the face it is obvious that pain is perceived, it may be assumed that the novocain injection has properly reached the nerve, and now another

whiff of anæsthetic is given, while the alcohol syringe is fitted on and the alcohol injected slowly drop by drop. This method I described in less detail in 1909, and on the whole it is very successful, though a skilled anæsthetist is advisable. The chief difficulty experienced is a liability for retching and vomiting to occur before the injection has been completed or the nerve has been found. Should this happen the needle should be withdrawn, and a fresh attempt made as soon as the vomiting is over.

Should the patient have been found to flinch equally when both sides are pricked, after the first novocain injection, no alcohol is injected, but the nerve again searched for as before, until the test shows it has been rendered anæsthetic by the novocain.

This method may be employed with success for any of the nerve trunks, though if the ganglion is to be injected it is best not to administer any more chloroform-ether after the third division has been anæsthetised by the novocain at the foramen ovale. There is very little pain produced by the subsequent slow injection of alcohol into the ganglion, and it is of the utmost importance to have the patient's conscious guidance as to the progress of the anæsthesia on the cheek and forehead.

If chloroform and ether anæsthesia is undesirable, a hypodermic injection of morphine and hyoscine may be used instead. A dose of a quarter of a grain of morphine with $\frac{1}{150}$th grain of hyoscine should be given half an hour before the time arranged for the nerve injection, the patient being kept quiet, with closed eyes and the room darkened to encourage drowsiness. By this means even a very nervous and apprehensive patient may be calmed sufficiently to bear the manipulations of the nerve injection without much flinching, and from this point of view it is almost an ideal method.

The patient's answers, however, to the tests by pin-prick for cutaneous anæsthesia after the injection of novocain or alcohol are likely to be much less reliable than when hyoscine and morphine have not been used. Hence there

is more liability to error through the patient giving faulty answers to tests for cutaneous anæsthesia, and it may be found later on that the nerve has not been properly injected.

My first preference, therefore, is local novocain only, but if the patient is very nervous, or a previous trial of injection has been found too painful, then the choice will lie between hyoscine-morphine and chloroform-ether anæsthesia, preferably the latter.

CHAPTER X

CASES RESEMBLING TRIGEMINAL NEURALGIA

Occasionally cases are met with which must be classed as chronic trigeminal neuralgia, and yet they differ from it in the most characteristic point, that the spasmodic attacks are not provoked by movements of eating or talking, or by handling the face. These patients therefore have no trigger zones, which is one of the most diagnostic points of trigeminal neuralgia. Two instances of this type will suffice.

Ten years ago I saw a man, *æt.* 65, who for six years had been suffering from spasmodic attacks of neuralgic tic in the left cheek and around the left eye and eyebrow. He said that the pain was sudden and flashing, mostly on the side of the nose and eyelids.

Some pain spread into the left upper gum, but never affected the lower jaw. For the first four years it was bearable, and he was usually free in the summer time, though a cold wind would bring it on. The pain then began to get worse, he having had only three days' freedom in the previous four months. During the attacks the left eye became bloodshot, but the neuralgia was not aggravated or started by eating, talking, or handling the face. Alcohol injection of the second division and afterwards of the third division and supra-orbital nerve gave him complete relief for five years. Recurrence then of the neuralgia decided me to attempt destruction of the Gasserian ganglion by injection. This I did in June 1919 successfully, and in 1924 he was still alive and well, with no recurrence of sensation over the trigeminal area, and no return of pain. Incidentally, it was remarked that he had had total loss of gustatory

sense on the left side of the tongue since the first injection of the foramen ovale.

A lady, *æt.* 66, whom I saw in 1917, had for fourteen years suffered from paroxysms of very severe pain in the left side of the temple and head which were not induced by eating, talking, or rubbing the face. The attacks were said to have been much worse since the previous year, when she had influenza and her husband died. During the seizures she would put her hands across the top of her head and writhe and scream with the agony. Injection of the Gasserian ganglion was performed through the foramen ovale by my ordinary route above the sigmoid notch. While this was being done she showed signs of the vestibular syndrome, intense vertigo with coarse nystagmus and vomiting, these symptoms, no doubt, being due to leakage of the alcohol on to the auditory nerve, either from the lateral recess, or backwards along the sensory root of the trigeminal; these symptoms cleared up in about twenty minutes, leaving fairly complete fifth-nerve anæsthesia. I heard in 1924 that she had been practically free from pain since this operation in 1917.

Auricular Neuralgia

In 1915 I saw a man, *æt.* 54, who for thirteen years had been subject to attacks of neuralgia in the right ear, which would develop within an hour or so after washing or rubbing the ear. The pain developed gradually for hours, working up to a sort of crisis of constant violent neuralgia in the ear, which would go dead white while the pain lasted. When the agony was at its worst he was unable to move at all until after the crisis, when the pain gradually disappeared. His only trouble was the sensitiveness of his left ear, as rubbing it would always start the neuralgia within an hour or two, and therefore of late years he left his ear severely alone, never washing it or rubbing it if he could help it. On examination the ear appeared quite normal except for accumulated dirt; there was no anæs-

thesia, no deafness, nor any abnormal physical signs. The most susceptible part of his ear was the concha, an area which is on the border line of cutaneous supply for the great auricular nerve from the cervical plexus, the auriculo-temporal branch of the fifth nerve and the sensory cutaneous fibres of the seventh or facial nerve. It is difficult to say to which nerve this neuralgia could be ascribed, but probably it should be included with the geniculate neuralgias (see p. 334).

Myoma Cutis associated with Paroxysmal Trigeminal Pain

In 1912 I saw, with the late Sir James Galloway, a man, *æt.* 34, who for the previous twenty years had noticed slightly raised reddish spots on his left cheek, mostly along the line of his lower jaw. Ten years previously to my seeing him these spots had been treated by electrolysis on twenty occasions, and a year or two later he noticed neuralgia commenced in the left side of his face on a cold bleak day. For some years he had pain on only one or two cold days, but latterly this had been becoming steadily worse, very intense for three or four winters, and daily during the last twelve months, though worse in a cold wind. The paroxysms were preceded by a stinging sensation in the left side of his cheek, which after a few seconds would develop into throbs of severe, biting, tearing pain, so intense that he could pay no attention to anything beyond his suffering, which might last about fifteen minutes. Emotion might start the pain, or even a minor chord of music, and at the commencement of the attack the reddish spots would rapidly change colour, becoming pallid and yellowish. When I told him I considered nerve injection would not benefit him I witnessed the rash rapidly fade to a yellow colour, followed by an attack of neuralgia. Sir James Galloway informed me that the spots were due to muscle fibres in the skin, the condition being known as myoma cutis, and

though the history of electrolysis being followed later by the neuralgia was suggestive of this being the primary cause of the attacks of pain, yet it is more than doubtful whether this was of any significance, as he had three similar spots on the front of his right thigh, where he also had the same kind of neuralgic pain if the weather was very cold, though it was less severe, and this area had been untreated.

Chronic Neuralgia of the Jaws

A more difficult group is that in which there is more or less persistent pain, though it may vary in severity, but the patient is scarcely ever completely free of pain affecting the region either of the upper or lower jaw on one side. Some of these cases appear to resemble the true trigeminal neuralgias in that the pain may be completely arrested by a successful alcohol injection of the second or third division of the fifth, according as to whether the pain affects the upper or the lower jaw, or else by a corresponding operation for avulsion of the infra-orbital nerve, or the Gasserian operation.

A girl, *æt.* 23, had suffered for two and a half years from pain in the right upper jaw. At first this was confined to the right upper bicuspid, but extraction of the tooth did not relieve the pain; the antrum was opened and washed out, and X-rays excluded any stump or impacted wisdom tooth. When I saw her in October 1909 the neuralgia was chronic and persistent, though apparently worse at times. In the right upper jaw only the incisors and upper canine tooth remained, the cheek was slightly tender and a trifle swollen over the antrum. Injection to the foramen rotundum failed to produce anæsthesia, and the pain was unrelieved until the infra-orbital nerve was twisted out by a surgeon from the floor of the orbit. Her pain then disappeared, and I heard of her years after as being quite well and doing good work in the War. Three years before I saw her, and before the facial neuralgia commenced, she had developed contracture of the right

third finger with anæsthesia of the hand, following a fall upon the right hand. There was still analgesia of the hand in 1909, and the right grip was weaker than the left, the probable diagnosis being hysterical contracture and anæsthesia. The question naturally arose, Was the facial neuralgia also of functional origin? Its cure after operation is, I think, weighty evidence against this, as functional neuralgias appear to become more fixed and inveterate after operation as a rule.

I saw a young lady, *æt.* 27, in 1923 for continuous neuralgia in the right upper gum as far as the middle line and down the lower jaw, which had commenced nine years before as a jumping pain in the right upper first bicuspid. The tooth was extracted, with no relief, and gradually all her teeth were removed, but still there was no improvement. Her pain had been continuous for the last two years, it was not made worse by eating or by movements of the face, but the neuralgia kept her awake, and on account of it she always ate on the left side.

Under chloroform anæsthesia I injected the third division at the foramen ovale, producing dense anæsthesia. The neuralgia disappeared from that time and her mother wrote three months later to say that all the old terrible pain had gone.

These chronic neuralgias of the jaws are almost restricted to young unmarried women, and this fact is naturally suggestive of an hypothesis of functional origin for the pain. Some cases, as those just related, have benefited by operation or by injection, while others apparently similar have failed to respond in any way to injection treatment.

Undoubtedly these cases are on a very different plane from ordinary spasmodic trigeminal neuralgia, and though injection is probably the best treatment and the patient should be encouraged to believe that a successful injection, preferably done under a general anæsthetic, is certain to relieve, yet the physician must be prepared to find no resulting improvement.

Other Persistent Pains in the Face

In addition to the forms of neuralgia already described persistent pains in the face may be due to posterior fossa tumours in the cerebello-pontine angle pressing upon the trunk of the fifth nerve, and cases have been recorded in which paroxysmal pain has persisted for years before the diagnosis was definitely established. Syringal gliosis and other intramedullary growths of the pons and medulla may cause trigeminal pain through involvement of the spinal descending root of the fifth; patches too of disseminated sclerosis may cause pains and paræsthesiæ of the face, associated with numbness and even partial anæsthesia, quite apart from genuine trigeminal neuralgia.

Thrombotic lesions in the pons and medulla also may involve the spinal root of the fifth, causing loss of sensation to pin-prick and to temperature, but no tactile anæsthesia on that side of the face. Sclerosis of this tract will result from such a lesion, and paroxysmal neuralgia over some part of the fifth nerve area may be a late sequel of this vascular lesion. The vessel which is most commonly involved by thrombosis in this region, causing softening of the side of the medulla, including the spinal root of the fifth, is the posterior inferior cerebellar artery.

A man, *æt.* 67, had a sudden "stroke," no loss of consciousness, but a sensation as if hot flannels had been suddenly applied to his head, and as though hundreds of needles were sticking into his scalp. At the same time he became intensely giddy and had to be put to bed. Two days later he became unable to swallow and required to be fed artificially for a week.

When I saw him six years later, in 1909, he was complaining of intermittent flashes of severe pain above the left temple and outside and below the left eye and cheek. The stroke had been followed immediately by slight aching round the eye and temple, which gradually increased in severity, developing into flashes of pain a year or two later, which darted through his left eye and temple towards

the inner canthus. Light touches on the skin anywhere on the left side of the face were liable to provoke a spasm of the neuralgia, in this respect resembling the trigger zones of chronic paroxysmal trigeminal neuralgia. He was completely analgesic to pin-prick, with loss of temperature sense over the whole left trigeminal area, including the tongue, but tactile sensation was practically normal, and there was no weakness of the masticatory muscles. There was crossed analgesia on the right side of the body, trunk, limbs, neck and back of head; that is to say, on everything below the trigeminal area on the opposite side a pin-prick was felt only as a touch, and warm and cold were not properly recognised on these areas. Tactile sensation, moreover, was not quite normal, though he was able to perceive light touches with cotton wool.

He was still shaky on his legs, especially if he stood on the left foot only, though his hands were perfectly steady. Eating was liable to start his pain, as also were light touches on the left side of the face, especially if anyone but himself were to touch it, and he was apt to flinch at once if anything approached his face, though pressure with the palm of his own hand appeared to give relief, and the pressure of his hat did not trouble him; the pain he described as a lightning flash gone in a moment or so.

No treatment seemed to give him any relief. The diagnosis in this case was perfectly clear, the sudden onset, intense vertigo and the subsequent crossed analgesia on one side of the face and of the limbs and trunk on the opposite side proving the lesion to be on the left side of the medulla, involving the left fillet and spinal root of the fifth. Although a great many cases have been recorded of thrombosis of the posterior inferior cerebellar artery, and I have myself seen a considerable number, probably thirty or more, I have not come across any other case in which paroxysmal neuralgia developed in the analgesic fifth nerve area, and it is the only case of the kind I have seen in which a symptom was described as of numbers of needles sticking into

the scalp. One other case I have seen which is comparable, but in which the lesion must have been at a higher level, probably in the neighbourhood of the principal sensory nucleus. In this case a gardener, *æt.* 42, had sudden pain in the right side of his face, which was still persistent three years later when I saw him in 1914. He described the pain as being like hot wires, and it varied in severity and was brought on by a hot sun or a cold wind. He had total paralysis of the whole of the right fifth nerve, motor and sensory, with loss of taste on the right half of the tongue. He had right ankle clonus and right extensor plantar reflex. The lesion in this case was probably a pontine thrombosis of syphilitic origin.

I have also seen persistent trigeminal neuralgia in three patients, all of whom had suffered from a fractured base from an injury involving the fifth nerve, probably in the neighbourhood of the Gasserian ganglion; in all three cases there was permanent fifth-nerve damage with motor and sensory paralysis, and in one, a woman who had received her injury through a severe blow on the right side of the forehead upon a door lintel, and in whom diplopia was present for the first three months, there was a typical Argyll Robertson pupil on the injured side, the only permanent result of partial third-nerve injury. She described her pains as stabbing in character, and they commenced about four and a half months after the injury. All these cases showed paroxysmal neuralgia of trigeminal distribution, sometimes associated with trigger zones, and aggravated by eating and handling the face.

Their superficial resemblance therefore to chronic paroxysmal trigeminal neuralgia might easily lead to confusion with this disease unless care were taken in the physical examination. The clue to the damage is, of course, the trigeminal anæsthesia, which is never present in any form in paroxysmal trigeminal neuralgia, unless operations on the nerve or alcohol injections have previously been done. Total fifth-nerve palsy is hardly likely to be overlooked, as the patient is sure to complain

of subjective numbness of the face, but in cases of lesion of the side of the medulla involving the spinal root of the fifth, and not the principal sensory nucleus, or the trunk of the nerve, the patients will not notice any loss of sensibility in all probability, unless they are specially observant, because tactile sensibility is not interfered with, and there is therefore no subjective sensation of numbness, and the loss of sensibility to pin-prick and to temperature will only be evident on careful testing.

I have already referred in the chapter on trigeminal neuralgia to the number of cases which I have met with of apparently typical chronic trigeminal neuralgia complicating disseminated sclerosis, about two per cent. of all my cases, and in several of these cases the facial neuralgia was bi-lateral (see pp. 158 and 160). Oppenheim,[1] in his text-book on "Diseases of the Nervous System," refers to one such case which he saw in which at the autopsy a patch of sclerosis was seen on the fifth nerve at its exit, but whether the exit of the nerve from the pons or from the skull is not stated. If the lesion causing this neuralgia is on the central side of the Gasserian ganglion it is difficult to understand how such cases can be relieved by alcohol injection performed at the foramina of exit of the nerve trunks from the skull. Moreover, in these patients there is never any anæsthesia to be detected over the trigeminal area in uninjected cases. The problem therefore of the association of apparently typical facial tic douloureux with disseminated sclerosis is a puzzling one (see pp. 394-5).

[1] Fifth ed. 1911, vol. i. p. 337.

CHAPTER XI

APPLIED ANATOMY AND PHYSIOLOGY OF THE FIFTH NERVE

TROPHIC FUNCTION OF THE FIFTH NERVE

Applied Anatomy and Physiology of the Fifth Nerve

The Gasserian ganglion is a small mass of unipolar nerve cells, about the size of a small bean, lying on the floor of the skull in the middle fossa, in a depression near the tip of the petrous bone, just above and internal to the foramen ovale, and adherent on its inner side to the outer wall of the cavernous sinus. Beneath the ganglion run the motor root of the fifth nerve and the great superficial petrosal nerve. On account of its shape this ganglion is sometimes called the semi-lunar ganglion, its convexity being forwards, and it is contained within a special sheath of dura mater known as the cave of Meckel, and is surrounded by cerebro-spinal fluid. To the concave back of the ganglion is attached the large sensory root of the fifth nerve, which, after leaving the pons, passes through a special channel in the dura mater above the ridge of the petrous bone, to enter the cave of Meckel. Tracing the sensory root of the fifth nerve backwards from the Gasserian ganglion, it enters the side of the pons on its upper surface, rather above the middle, where it passes inwards to reach the upper sensory nucleus of the fifth nerve, a hemispherical mass of grey matter known as the convolutio trigemini, situated outside and ventral to the motor nucleus. Before reaching this nucleus the fibres of the sensory root bifurcate, and those fibres which carry tactile and pressure sensations terminate in the nerve cells of this nucleus.

New fibres carrying these sensations upwards to the

thalamus pass inwards from the nucleus as the raphé bundle, and decussate in the middle line to form the trigemino-thalamic tract, or trigeminal fillet, which keeps separate from the remainder of the fillet fibres and ends in the lateral nucleus of the thalamus.

A lesion of the pons at the level of the upper sensory nucleus of the fifth nerve will be likely to damage cutaneous sensations over the trigeminal area, but not those for pain and temperature. Thus, Spiller [1] described a case of syringomyelia involving the main sensory nucleus of the fifth, but not the nucleus of the descending spinal root. Tactile impressions were abolished but sensation to pain was normal. I have seen precisely similar dissociated anæsthesia of one trigeminal area, with bi-lateral loss of taste on the front two-thirds of the tongue, in a case of glioma of the lower crus and upper part of the pons.

After the bifurcated fibres of the sensory root have given off the small branch which runs dorsally to end in the main sensory nucleus, the principal branch turns downwards at a right angle, the bundle sweeping through the pons and the medulla in a tract or nerve bundle known as the spinal or descending root of the fifth nerve. These nerve fibres end in nerve cells of the substantia gelatinosa of Rolando adjacent to this tract, which can be traced downwards as far as the second cervical segment of the cord. New fibres from this mass of grey matter, which may be called the nucleus of the spinal root of the fifth nerve, carry the pain and temperature sensations upwards by decussating in the middle line and joining the opposite fillet. A lesion therefore of the side of the medulla or lower part of the pons, by damaging the spinal root of the fifth, will at the same time probably injure the mesial fillet of the same side and the inferior cerebellar peduncle, so that a well-known clinical syndrome is produced, in which the prominent symptoms are intense ataxy and vertigo, with analgesia and thermanæsthesia of the trigeminal area on the same side of the head, together

[1] *Jour. Nerv. and Ment. Dis.* 1915, Vol. 42, p. 399.

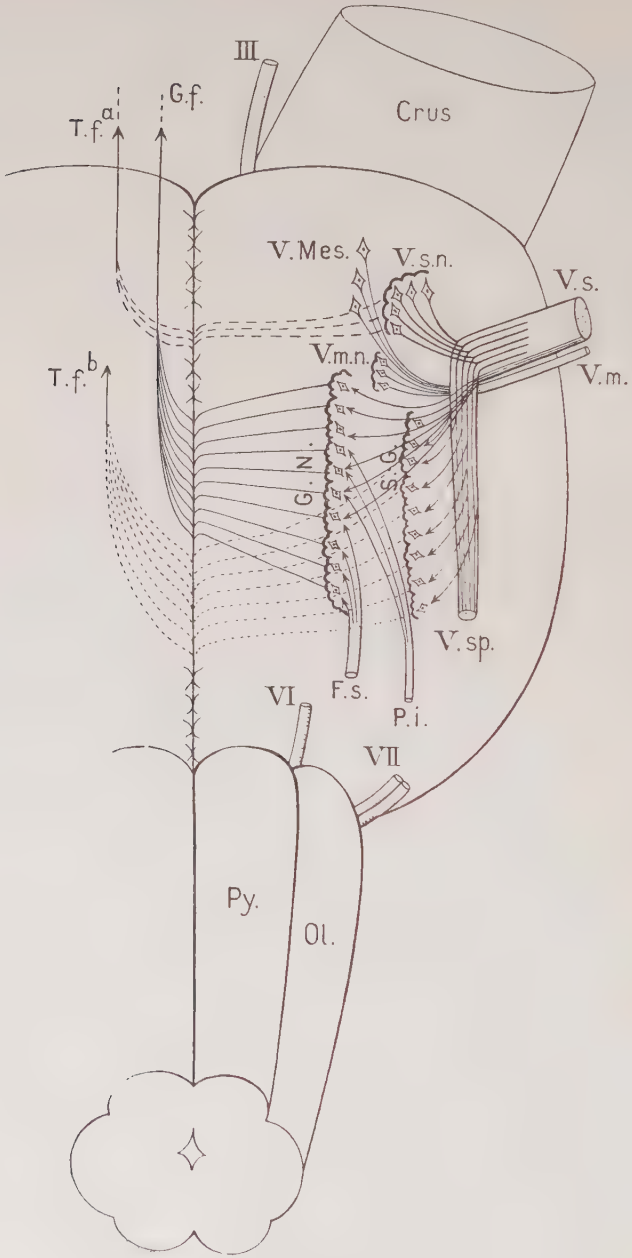

Fig. 35.

with analgesia and thermanæsthesia of the opposite side of the body below the fifth nerve area. One of the common causes of this clinical picture is thrombosis of the posterior inferior cerebellar artery of one side, a small vessel arising from the vertebral artery just before it joins its fellow to form the median basilar artery.

The Gasserian ganglion gives off three main branches, each of which has a small ganglion in connection with it. The ophthalmic and maxillary branches are entirely sensory in function, the motor root of the fifth nerve being distributed with the mandibular nerve or third division only.

The first or ophthalmic branch lies closely attached to the outer wall of the cavernous sinus, and passes forwards to leave the skull by the sphenoidal fissure, where it divides into frontal, nasal and lachrymal branches. It supplies the eyeball with sensation through the ciliary branches of the nasal nerve, the skin and mucous membrane of the upper eyelid, and the cornea and the rest of the conjunctiva, with the exception of that lining the lower lid. It is said that the corneal supply contains no tactile fibres but only those serving the sensation of pain. It is still debated whether there is any special *trophic function* in the nerve supply to the cornea. In some cases

FIG. 35.—Diagram of ventral view of the intra-pontine connections of the left fifth nerve, to illustrate the path of the taste fibres.

V.s.	Sensory trigeminal root.
V.m.	Motor trigeminal root.
V.s.n.	Principal sensory nucleus of fifth nerve, or convolutio trigemini.
V.m.n.	Motor nucleus of fifth nerve.
V.Mes.	Mesencephalic nucleus of fifth nerve.
V.sp.	Spinal root of fifth nerve.
S.G.	Substantia gelatinosa or nucleus of spinal trigeminal root.
G.N.	Gustatory nucleus.
F.s.	Fasciculus solitarius, or ascending glossopharyngeal root.
P.i.	Pars intermedia Wrisbergi.
Py.	Pyramid.
Ol.	Olive.
T.f.a.	Trigeminal fillet (tactile and pressure).
T.f.b.	Trigeminal fillet (pain and temperature).
G.f.	Gustatory fillet.

destruction of the Gasserian ganglion or its sensory root is followed by no signs of ophthalmic disturbance, but in others, even though the eye be carefully kept closed and irrigated with boracic lotion, conjunctivitis with uveal injection and widespread loss of corneal epithelium appear within three or four days, which may spread to the deeper layers of the cornea, a neuro-paralytic keratitis.

Those who have had many of these cases to treat find it hard to resist the theory of a trophic supply to the cornea, and in this respect an analogy may be drawn with the bladder and the impossibility of preventing the development of cystitis in certain cases of inflammatory and irritative lesions of the lumbo-sacral cord.

The cutaneous branches of the first division supply the skin of the eyebrow and forehead as far back as the vertex, through the supra-orbital and supra-trochlear nerves, and the upper side of the nose from the inner canthus down to the tip. Branches from the frontal division of the ophthalmic nerve also supply the mucous membrane lining the frontal sinus, the ethmoidal and sphenoidal sinuses receiving fibres from the nasal branch.

Catarrhal inflammation of the frontal sinus is therefore liable to cause referred pain over the supra-orbital nerve, and tenderness at the supra-orbital notch, and it is quite common for a sufferer from an influenzal catarrh to complain of hyperæsthesia of the forehead and front of the scalp when using a hair-brush or comb.

The *ciliary ganglion* is connected with the nasal nerve, having also a sympathetic root, and a motor root from the branch of the third nerve to the inferior oblique muscle. Neuritis of the ciliary ganglion therefore causes weakness of accommodation and sluggish pupil reaction, with intense pain in the eyeball, and diplopia probably due to involvement of the inferior oblique branch.

The second division or *maxillary nerve* leaves the Gasserian ganglion to pass forwards nearly half an inch to the foramen rotundum, a circular foramen in the sphenoid bone situated about a quarter of an inch below the rim of

the sphenoidal fissure. Before entering the foramen the nerve gives off two fine recurrent *meningeal branches*, which communicate with similar meningeal branches from the third division, and supply the dura mater of the anterior half of the head. The first or ophthalmic division also gives a recurrent meningeal branch which supplies the tentorium.

These meningeal branches of the fifth nerve have a special clinical significance in that it has been observed that certain types of non-migrainous headache never recur on the operated side after destruction of the Gasserian ganglion. It is probably headaches of rheumatic type, from chill, and from certain reflex causes, such as eye-strain, that are thus cured by destruction of the dural meningeal nerve supply. Conversely, affections of the meninges, such as serous meningitis and migraine, may set up reflex pains in the teeth and face. For example, a lady when *æt.* 23 had a sudden attack of hemianopia while at breakfast, lasting half an hour and succeeded by violent headache, the pain running along the jaws into her teeth so that she felt certain that she had dental trouble and sought her dentist, as the pain in her face had lasted three days. Her dentist, however, assured her that her teeth were sound, and the facial neuralgia soon disappeared, being clearly of reflex origin through the meningeal trigeminal branches, which were irritated by the migrainous vaso-motor spasm.

After emerging from the foramen rotundum into the spheno-maxillary fossa behind the orbit, the maxillary nerve gives off the temporo-malar branch which supplies the skin over the malar bone and also the temple and side of the forehead. These are two very common areas for referred pain in neuralgia affecting the second division, but destruction of the maxillary nerve produces no anæsthesia of the temple beyond the outer canthus of the eye, owing to the overlapping supply by the auriculo-temporal and supra-orbital nerves. In the spheno-maxillary fossa the second division also gives two sensory branches to the

spheno-palatine or *Meckel's ganglion*, which lies close to the spheno-palatine foramen. This ganglion is said to be anæsthetised by cocainisation of the mucous membrane at the upper posterior portion of the middle nasal fossa (see Sluder's neuralgia).

The main sensory branches of this ganglion are the palatine nerves, the two largest of which are described as direct from the maxillary nerve, merely passing through Meckel's ganglion to supply the gums and mucous membrane of the hard and soft palate. They descend from Meckel's ganglion through the posterior palatine canal, which opens at the posterior and outer angle of the hard palate, opposite the last molar tooth. With the mouth wide open, it is possible to pass a needle up this canal, and thus to inject Meckel's ganglion directly with alcohol, though it is not a treatment worth practising.

The nasal branches of the ganglion enter the nasal fossæ by the spheno-palatine foramen, and supply with sensation the mucous membrane of the nasal fossæ and septum. Meckel's ganglion is also connected by the Vidian nerve and the great superficial petrosal nerve to the geniculate ganglion on the facial nerve, but the function of this communication is doubtful. When the maxillary nerve is injected with alcohol in the spheno-maxillary fossa the needle may hit the nerve either behind or in front of its branches to Meckel's ganglion. Consequently, in some cases of successful injection of the nerve the palate is not anæsthetised, although the teeth, gums and cheek may be deeply anæsthetic.

The maxillary nerve next gives off the posterior and middle dental branches to supply the three molar teeth and the bicuspids, and then enters the infra-orbital canal, and shortly before its exit from the infra-orbital foramen the anterior dental branch is given off to supply the canine and incisors.

For this reason alcohol injection of the infra-orbital nerve is sometimes most successful in arresting a chronic trigeminal neuralgia of the second division, as the needle

can be pushed some little distance along the infra-orbital canal, and the anterior dental branch included in the injection. On the other hand, injection of the mental foramen is nearly always valueless in arresting a chronic mandibular neuralgia.

The infra-orbital nerve issues a quarter of an inch below the rim of the orbit, and one inch from the middle line, the three foramina, supra-orbital, infra-orbital and mental, being almost in the same vertical line. The nerve is covered at first by the levator labii superioris muscle; hence alcohol injection of the infra-orbital nerve may cause weakness in raising the upper lip.

The cutaneous supply of the nerve is distributed to the cheek and upper lip, with its mucous membrane, and the side of the nose. The anæsthesia produced by destruction of the infra-orbital nerve is bounded externally by a line adjoining the external canthus to the angle of the mouth, and internally at the inner canthus of the eye.

The third and largest of the three divisions of the fifth nerve plunges downwards at once from the ganglion into the foramen ovale, an oval opening in the great wing of the sphenoid, which looks downwards and slightly outwards and forwards. The small motor root emerging from beneath the ganglion passes through the foramen ovale with the third division, its fibres forming a partial network with the sensory fibres, known as the plexus of Santorini. As soon as the third division has passed through the foramen ovale it gives off a recurrent meningeal branch which re-enters the skull with the meningeal artery through the foramen spinosum.

The foramen ovale is the point of exit through which the third division of the fifth nerve leaves the skull, and is an oval opening in the great wing of the sphenoid bone immediately outside and below the edge of the Gasserian ganglion. Close behind it is the foramen spinosum, through which the middle meningeal artery enters the skull, after passing between two branches of the third division which join to form the auriculo-temporal nerve.

From this nerve is given the sensory branch to the *otic ganglion*, which communicates with the geniculate ganglion on the seventh nerve by means of the small superficial petrosal nerve. Probably no muscular motor nerve fibres pass through the otic ganglion. The auriculo-temporal nerve supplies a racquet-shaped area of skin on the temple and side of the head above the level of the incisura notch, communicating in front with the supra-orbital nerve and the temporo-malar branch of the second division. The auriculo-temporal nerve also supplies the anterior portion of the pinna and the anterior wall of the external auditory meatus and the outer wall of the tympanum in part. In addition to the auriculo-temporal nerve the third division gives two other main branches, the inferior dental and lingual nerves, and also the buccal branches to the side of the cheek. The inferior dental nerve is the largest of the three main branches, and passes forwards and downwards to enter the mandible by the inferior dental foramen, which is guarded on its inner side by the spine of Spix. At this point the nerve is frequently injected with novocain by dentists to produce anæsthesia of the lower jaw for dental fillings, etc. The nerve may also be injected here with alcohol in cases of chronic mandibular neuralgia. Supplying the teeth of the lower jaw, the nerve emerges at the mental foramen opposite the second bicuspid tooth to supply cutaneous sensibility to the chin and lower lip.

The lingual nerve supplies one-half of the anterior two-thirds of the tongue with ordinary sensation, being joined by the chorda tympani after the latter has left the facial nerve in the middle ear. The lingual nerve is generally believed to carry no taste fibres, which reach it by the chorda tympani, and consequently section of the lingual nerve behind this point abolishes sensibility on the front of the tongue to tactile and painful impressions, but may leave taste unimpaired. Pressure pain upon the tongue is thought by some to be supplied by nerve fibres running with the chorda tympani or in the hypo-glossal nerve, which is the motor nerve to the tongue, just as it is argued

that pressure pain sensation on the face is supplied by sensory branches of the facial nerve, and not by the trigeminal. I am somewhat sceptical of this theory that pressure pain is a function of nerve fibres accompanying the motor nerve to the part, such as the facial or the hypoglossal, as I have on many occasions seen total loss of all sensation, including hard pressure, as a result of ganglion injections or of injections of the separate branches, but it is difficult for me to speak with any certainty on this point, as in numerous other cases, in which I have found ordinary touch and pin prick abolished but sensibility to deep pressure present, the retention of the latter form of sensation may have been due to incompleteness of destruction of all the nerve fibres by the injection. This point could only be settled by careful examination in such a case of a series of complete sections of the sensory root of the Gasserian ganglion. Maloney and Foster Kennedy,[1] in their work on gasserectomies, found that ordinary pressure touch is carried by the fifth nerve, and that pressure pain persists in the eye. According to those authors the facial nerve contains no pressure sense fibres distal to the Fallopian canal. On the other hand, Davis,[2] investigating cases following Gasserian operations, concludes that deep pressure pain persists over the face, the fibres running in the facial nerve and in the nerve of Wrisberg. This seems to me a more likely course to explain the return of some deep pressure sense after Gasserian destruction, though there can be little doubt that the trigeminal nerve conveys ordinary pressure touch sensation to the whole face, jaws and tongue.

The *motor root* after leaving the skull by the foramen ovale supplies motor fibres to all the muscles of mastication, the temporal, masseter, internal and external pterygoids, mylohyoid and the anterior belly of the digastric. It was formerly said also to supply the tensor tympani, but this is probably not true, nor does it supply any motor fibres to the palate. The buccinator muscle

[1] *Brain*, 1911, Vol. 34, pp. 1–28.
[2] *Arch. for Neur. and Psych.* 1923, p. 283.

receives its motor supply from the seventh nerve. The motor root arises from a group of nerve cells in the pons situated above and internal to the principal sensory nucleus of the fifth nerve. Occasionally there is a communication between the motor nucleus and the third nucleus, especially between that portion supplying the depressors of the lower jaw with the nerve to the levator palpebræ. The effect of this is to produce the phenomenon described first by Gunn [1] known as "jaw-winking," so that the action of the external pterygoid upon opening the mouth wide causes a retraction of the upper eyelid on the same side. There is always slight ptosis from congenital weakness of the levator palpebræ in these persons.

Joining the motor root on its way through the pons is a small band of fibres originating from scattered nerve cells above the motor nucleus. This is known as the mesencephalic root, and according to May and Horsley [2] contains both efferent and afferent fibres. It has been stated to be motor in function, and to supply the depressors of the mandible. The cells of origin of this mesencephalic root lie much nearer the third nucleus than does the principal motor nucleus of the fifth nerve, and jaw-winking is easier to understand if these fibres supply the depressors of the lower jaw.

Trophic Functions of the Fifth Nerve

The fifth nerve has certain influences on nutrition besides that of the motor root on the masticatory muscles. It has often been noticed that persons who are subject to frequent neuralgic frontal headaches on one side may develop unilateral *greyness of the hair* in front, as far inwards as the line of the supra-orbital nerve at the notch. Hilton described the case of a man who had a painful carious lower molar tooth, which caused greyness of the hair on the temple of the same side only, so that the man had his head shaved and wore a wig to hide it. In the case of a woman suffering from morphœa, with left *facial hemiatrophy* and

[1] *Trans. Ophth. Soc.*, 1883, Vol. 3, p. 283.
[2] *Brain*, 1910, Vol. 33, p. 175.

trigeminal neuritis, mostly of the first division and to a less extent of the second branch, the left eye was sunken and the left frontal bone and maxilla were much wasted, though not as far as the mid line of the face, ending at the level of the supra-orbital notch. Parallel with this line the scalp was bald on the left side for a wide streak running back to the vertex, with the skin atrophic and glossy (see Fig. 39, p. 283). A noteworthy point is that the trophic loss described

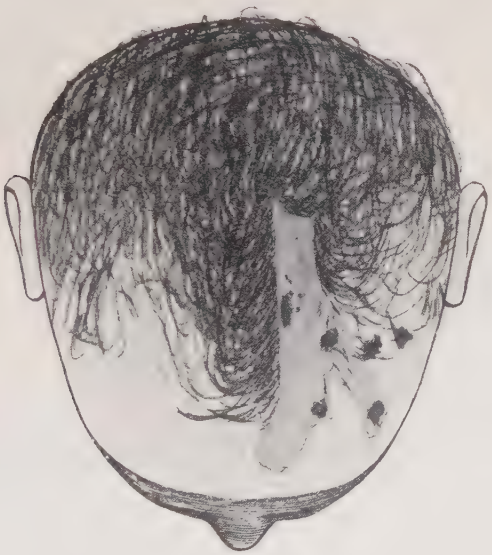

Fig. 36.—View of top of head showing trophic loss of hair and blebs due to irritant neuritis in case of gumma of left fifth nerve.

of greyness, baldness and atrophy of the bones of the face and the skin, does not extend as far inwards as the middle line, but ceases about the line of the supra-orbital notch.

The same point is to be noticed in the case of a man whom I saw in 1896 under the care of my former chief, Sir William Gowers.

A groom, *æt.* 46, had suffered ten months previously with pain for six weeks in all the teeth of the left side. This was followed by left-sided fronto-temporal headache and numbness of the left side of the face and mouth. Four

months later weakness of the right lower face developed, and a few weeks after herpes frontalis appeared on the left side, with falling out of the hair in that region. Two days before admission to hospital weakness of the right hand and arm appeared. There was then almost total anæsthesia of the left trigeminal distribution, with motor palsy and complete loss of taste on the left anterior two-thirds of the tongue.

There was a band of complete alopecia on the left side of the front of the head, with scattered scars of zoster vesicles, still bleeding, which the man asserted was due to

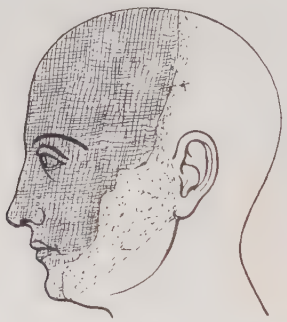

Fig. 37.—Area of complete and partial anæsthesia of face and head in the same case as Fig. 36.

scratching. This statement of the man's gave rise to an amusing error on Dr. Gowers's part, a physician of unusually brilliant clinical acumen. He had seen the man in private consultation and sent him direct to the National Hospital for one of his Wednesday lectures. He demonstrated the case to a large class the same day as a tumour of the left cortical facial centre, having quite overlooked the left fifth nerve lesion, being afraid to touch the man's head and pointing out to the class the left patch of alopecia and the bleeding herpetic scars as the result of scratching due to pediculi capitis ! (Fig. 36).

On his next visit to the hospital I was blamed for not having told him of the fifth nerve lesion before ! It was probably a gumma on the trunk of the fifth nerve pressing

on the side of the pons, and causing facial paresis of cerebral type on the other side. All the symptoms cleared up under the administration of mercury and iodide of potassium.

Sweat secretion on the face and forehead appears to be influenced both by lesions of the cervical sympathetic and of the trigeminal nerves.

In old-standing paralysis of the cervical sympathetic it is frequently noticed that in addition to slight ptosis and small pupil on that side there is also diminution or loss of sweating. This may sometimes be demonstrated in a man by examination of his hat lining, which will be much less stained on the affected side. Excessive sympathetic action probably causes increased perspiration, a phenomenon which is common in Graves's disease, in which excessive sympathetic action is probable.

On the other hand, lesions of the trigeminal nerve may be associated with marked hyperidrosis of the face. This I have seen in chronic supra-orbital neuritis, also as a sequel of trigeminal herpes, and in syringomyelia. In the two latter forms the sweating may be paradoxical in type, increased in cold weather, and by eating pungent or aromatic substances.

Supra-orbital Neuritis.—A man, *æt.* 51, had for some weeks complained of severe daily pains, lasting for five to six hours at a time, in the left side of the forehead and upper jaw. During the painful attacks he noticed great perspiration on the affected area of the forehead and side of the nose. Examination revealed anæsthesia of the left supra-orbital nerve area only, excluding the supra-trochlear and other branches of the first division of the fifth. The sinuses were clear, and no cause could be found for the neuritis, but an interesting point was the evidence of cervical sympathetic lesion, by the presence of slight ptosis and contracted pupil on the same side.

Post-herpetic Trigeminal Hyperidrosis.—This may be profuse, and is liable to be aggravated by eating, or by nervousness. (See chapter on Herpes, p. 291.)

Lachrymation and Salivation are frequent accompani-

ments of the neuralgic spasms in chronic trigeminal neuralgia affecting the second and the third divisions. Lachrymation is also profuse in the neuralgic ocular attacks in tabes, known as Pel's crises, in which the pain and flow of tears are accompanied by conjunctival swelling and redness.

Neuro-paralytic Keratitis has already been discussed in the chapter on ganglion injection. Its occurrence after certain cases of Gasserian operation or injection and severe ophthalmic zoster is suggestive of irritation within the ganglion being the cause of the keratitis. At the same time zoster vesicles may appear on the lips and chin after injection of the foramen ovale, though never after injection of the foramen rotundum.

Chronic neuritis of the trigeminal may cause glossy skin in morphœa, and hemiatrophy of the tissues, while the hair may fall out and show greyness from chronic irritation, as in gumma, neuritis and recurrent headaches. If all irritation is absent, a section of the sensory trigeminal root or destruction of the ganglion produces no visible trophic disturbances of skin, hair, cornea or other tissues.

CHAPTER XII

PATH OF THE TASTE FIBRES

The spinal root of the fifth nerve without doubt carries impressions of pain and temperature, and probably in its upper portion also sensations of taste, for Nageotte[1] has shown that some fibres from the trigeminal spinal root terminate in the upper part of the gustatory nucleus, where they blend with the upper fibres of the pars intermedia Wrisbergi. This gustatory nucleus is continuous below with the substantia gelatinosa of the solitary bundle, or ascending glosso-pharyngeal root; it measures fourteen millimetres in a vertical direction, beginning two millimetres below the main sensory nucleus of the fifth nerve, and ending three or four millimetres below the origin of the solitary bundle. In its uppermost portion this gustatory nucleus receives fibres from the spinal root of the trigeminal nerve only, and trigeminal fibres can be traced also in the median portion of the nucleus in communication with the pars intermedia Wrisbergi. Below the nucleus receives glosso-pharyngeal fibres, and over its median eight millimetres the fibres of the pars intermedia Wrisbergi terminate. Nageotte considers all these three branches of the fifth, seventh and ninth cranial nerves are gustatory in function.

For fifty years or more the anatomical path for taste impressions has been a matter of controversy. Forty years ago Gowers asserted that taste was entirely a function of the fifth nerve, and he published several cases of fifth nerve paralysis with total loss of taste, both on the front and back of the tongue. Erb also considered that taste

[1] *Rev. of Neur. and Psych.* Edinb. 1906, Vol. 4, p. 473.

was a function of the fifth nerve; Krause, however, a pioneer of Gasserian operations, came to the conclusion that the glosso-pharyngeal was the only nerve of taste, as in six cases of gasserectomy he found taste abolished in two, impaired in three, and unaffected in one case. On these grounds he argued that the trigeminus contains no taste fibres, but that taste is carried by the glosso-pharyngeal nerve, an argument which from his data seems difficult to follow. Cushing, on the other hand, concludes from his examination of cases after Gasserian operation that though in practically all there is either complete abolition or blunting of taste as an initial result, yet in the large majority there is complete return of taste. He suggests that the temporary loss of taste may be due to descending effects of nerve degeneration following the operation.

Morriston Davies[1] personally examined twenty cases of gasserectomy, and in only one did he find complete loss of taste, two and a quarter years afterwards, in this case taste being lost both on the front and back of the tongue and on the palate. In several of his cases, however, he found early loss of taste, which subsequently recovered.

Cushing and most modern authors argue on these grounds that the fifth nerve is not a gustatory nerve, but that taste sensations are carried from the tongue in front by the chorda tympani as far as the geniculate ganglion, and thence onwards to the pons by the pars intermedia, or nerve of Wrisberg, which may be looked upon as the sensory root of the seventh nerve, the motor nerve of the face. Developmental study and comparative anatomy point to the geniculate ganglion as being a sensory posterior root ganglion, homologous with the Gasserian and other spinal root ganglia, the seventh nerve or portio dura being comparable to the motor root of the fifth, or to an anterior spinal root, and the nerve of Wrisberg representing the posterior sensory root. These anatomical points are therefore in favour of the nerve of Wrisberg being the central continuation of the chorda tympani, and therefore

[1] *Brain*, 1907, Vol. 30, p. 219.

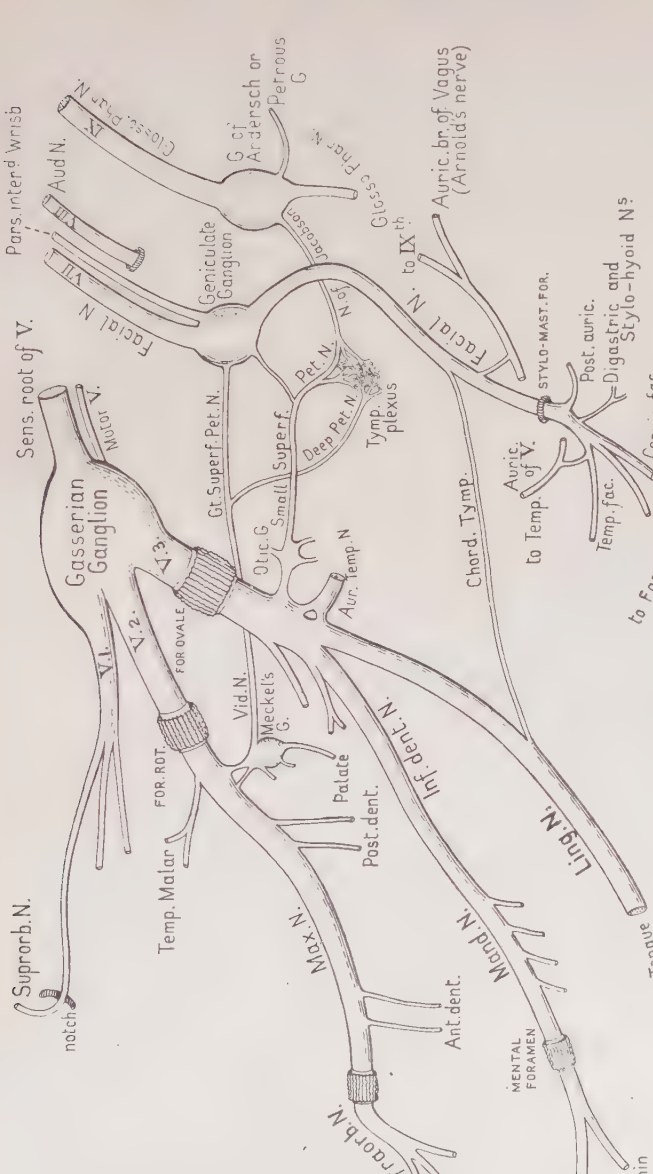

FIG. 38.—Diagram of trigeminal nerve connections with the facial, glosso-pharyngeal and vagus nerves.

being a nerve of taste. If this were the only path for conveying taste impressions from the anterior two-thirds of the tongue, then taste would be lost permanently in all lesions of the nerve of Wrisberg. Lesions of its peripheral portion, that is to say, the chorda tympani, are commonly accompanied by loss of taste in the early stages, as in acute facial paralysis or Bell's palsy, where this is of the ordinary type, or due to middle ear suppuration. I have seen and demonstrated loss of taste in early cases of Bell's palsy many scores of times, and I have also seen permanent loss of taste many years after destruction of the facial nerve by an extensive mastoid operation, with no motor recovery or facial contracture.

I have also found loss of taste on one side of the tongue in a case of geniculate herpes without facial palsy (p. 318).

Lesions of the nerve of Wrisberg are very much rarer and more difficult to prove, but in those cases which I have been able to test of fractured base involving the seventh and eighth nerves I have never found loss of taste. I have examined three cases of double facial paralysis with total nerve deafness, due to symmetrical syphilitic neuritis of the seventh and eighth nerves, and in none of the three was there any loss of taste, though, as both the facial paralysis and the deafness were total, it seems probable that the nerves of Wrisberg were also involved, but this is presumption only and not proof.

I have, however, seen total loss of taste on the front half of the tongue, with retention of normal acuity of taste on the back of the tongue, as a result of an intracranial alcohol injection of the inner two-thirds of the Gasserian ganglion. Leakage of alcohol had occurred through the ganglion into the lateral recess, producing the "vestibular syndrome" of violent nystagmus, vertigo and vomiting, with facial nerve paralysis. No deafness was produced, the lesion apparently being limited to the facial nerve, pars intermedia Wrisbergi, and the vestibular portion of the auditory nerve. The alcohol injection was done for severe long-standing post-herpetic neuralgia of

the ophthalmic division of the fifth nerve, and I therefore injected only the inner portion of the Gasserian ganglion with alcohol. As a result there was total anæsthesia of the whole ophthalmic division, including the eyeball, nearly complete anæsthesia of the second division, upper jaw, cheek and palate, but no trace of anæsthesia on the lower lip or tongue, the mandibular portion having escaped damage, as I had intended. It was clear therefore that the loss of taste on the front half of the tongue must be due to the pars intermedia Wrisbergi having been involved by the alcohol leakage. It could not be due to alcohol entering the middle ear via the Eustachian tube, and thus damaging the facial nerve and the chorda tympani, as there was no trace of deafness produced, and the Gasserian destruction proved the injection to have been within the foramen ovale intracranially.

In 1909 Pierce Clarke and Taylor in America published a case of severe tic douloureux of the seventh nerve, or geniculate neuralgia, in which were divided intracranially the motor seventh, the nerve of Wrisberg, and the upper fibres of the eighth nerve. In the published full report of this case no reference whatever to the condition of taste sensation is made, which is unfortunate, as the case was a unique opportunity for testing the gustatory function of the pars intermedia Wrisbergi. There was no return of the neuralgia six years later, and the absence of cutaneous anæsthesia was commented on, together with return of hearing after ten days, and recovery of the facial muscular power.

There seems little doubt that taste perception is possible on areas of the soft palate which are supplied with sensation by the second division of the fifth nerve, and unless these gustatory impressions are carried by the fifth nerve to the brain it would be necessary to explain these sensations of taste as being due to the glosso-pharyngeal nerve supplying gustatory sensation further forward than its area for tactile and painful stimuli. It is true that Ramsay Hunt figures a few herpetic vesicles on the soft palate in cases of

geniculate zoster, indicating a vestigial remnant of supply by the sensory fibres of the seventh nerve to this part, and I have myself seen a case of geniculate herpes with facial palsy accompanied by an outcrop of zoster on the same side of the soft palate and along the side of the tongue (p. 321). Such palatal taste fibres may perhaps reach the geniculate ganglion through the palatine nerves, Meckel's ganglion, the Vidian and great superficial petrosal nerves. and it is an attractive theory to consider the pars intermedia and the geniculate ganglion as the path for taste sensations from the tongue and palate, through the chief branches of the geniculate ganglion, the chorda tympani and great superficial petrosal nerves.

Experience in testing large numbers of cases in which taste sensation is abolished or impaired in lesions of the fifth, seventh or ninth cranial nerves leads me irresistibly to the conclusion that there is a correlation and interdependence of function as regards taste in all three of these nerves, and that a lesion of any one of the three may, and generally does, impair the function of taste on that side. Particularly is this interdependence of function noticeable with the fifth and seventh nerves, and comparative anatomical considerations are the probable clue to this fact. The seventh cranial nerve is the nerve of the first gill slit, and the lower we trace in the biological tree the comparative importance of the fifth and seventh cranial nerves the less important does the trigeminal nerve appear, while the sensory portion of the seventh nerve is definite, as in the Selachia, or sharks and rays, which are amongst the most primitive of vertebrates. As we ascend the scale of development towards mammals and the human species, so does the face become more and more important as the setting for the eyes and their increasing function of binocular stereoscopic vision, and particularly as the organ of display of the emotions through the facial muscles. With this gradual increase in functional development of the face so does the sensory portion of the trigeminal nerve acquire importance at the expense of the sensory portion of

the seventh nerve, which in man and the higher mammals is reduced to vestigial remnants, with the exception of the chorda tympani.

It should therefore not be a matter for surprise that varying results obtain in different individuals, that total loss of taste is present both on the front and the back of the tongue in some individuals in lesions limited to the fifth nerve, while in others it may be only impaired, or even not noticeably affected. The gustatory sense may thus in some cases belong entirely to the fifth nerve, while in others the loss of tactile sensation on the tongue may at first prevent gustatory stimuli from the chorda tympani from arousing a sensation of taste. This is the group in which taste is at first completely lost in fifth nerve lesions, but recovers, partially or completely, after a varying interval, though fifth nerve anæsthesia persists. In a third group in which taste is unimpaired on the tongue in spite of total trigeminal anæsthesia, the chorda tympani is sufficient alone for carrying full gustatory impressions.

The territory and function of the glosso-pharyngeal nerve are less subject to variation, and though in a few undoubted instances taste is totally lost on the back of the tongue in fifth nerve lesions, yet in the large majority of cases taste sensations on the back of the tongue and fauces are carried by the glosso-pharyngeal. These two nerves overlap each other in their sensory supply to the back of the tongue and fauces, they both carry gustatory impressions, they are the two sensory nerves concerned in the act of eating and deglutition, and they alone of all the nerves in the body, with the possible exception of the sensory fibres of the seventh nerve, are subject to an identical type of paroxysmal neuralgia, or tic.

My clinical experience of fifth nerve paralysis, both due to disease and when produced by injection of the nerve at the foramen ovale, is, in my opinion, indubitable evidence of the gustatory function of the trigeminus.

I have already described (p. 244) the case of a man suffering from a gumma on the root of the fifth nerve,

pressing on the pons, in whom taste was completely lost on the anterior two-thirds of the left side of his tongue, though the gustatory sense was quite acute on the right side. No other cranial nerve was affected, and the man made a complete recovery under iodide of potassium.

A man, *æt.* 31, developed a cerebral gumma causing total paralysis of the left fifth nerve, motor and sensory. The anæsthesia extended over the hard and soft palate as far back as the anterior pillar of the fauces, but there was no anæsthesia of the tonsil or of the pharynx. No other cranial nerve at first was involved, but eighteen months later he developed double third nerve palsy. There was total loss of taste on the left side, front and back of the tongue, and on the left side of the palate, to sweet, salt and bitter.

A woman, *æt.* 50, had total paralysis of the right fifth and sixth nerves with slight weakness of the right seventh, eighth and ninth nerves, as shown by partial weakness of the right side of the face, with twitchings of the face, slight right-sided deafness and partial anæsthesia of the right side of the pharynx. She was able to drink very hot fluids if she was careful to keep the liquid on the right side. The fifth nerve palsy was probably of sixteen years' duration, as at that time she had pain in the head followed by numbness of the right side of the face, of sudden onset. She never recovered the sensation of the face, and formerly she used to bite the tongue, and she had lost the vision in the right eye from corneal ulcer. Taste sensation was entirely lost on the anterior two-thirds of the right half of the tongue, but quinine was readily tasted on the right side at the back, though less strongly than on the left side. She had no other physical signs except right-sided loss of smell, no weakness of arm or leg, though of late years she had been becoming somewhat unsteady in walking.

A married woman, *æt.* 40, when five months pregnant, fell, receiving a violent blow on the right side of her forehead, being stunned for some minutes. Four and a half months later she developed severe pain over the right side of her

forehead and cheek, for which she had most of the teeth out on the right side. Partial right third nerve paralysis also developed. Nine months later she had recurring convulsions, with vomiting, for six weeks. Ever since she has had constant pain in the face, and when examined three years later there was complete sensory paralysis of the right fifth nerve, the anæsthesia extending back to the soft palate. Taste was completely lost on the right side of the tongue, both front and back, tested with quinine and sugar. There was a scar on the cornea from ulceration, and the right pupil was very sluggish, the only remaining evidence of the third nerve paralysis. There was no sign of syphilis, and the lesion was probably fracture of the base of the skull through the middle and anterior fossæ, causing pachymeningitis. She had a constant discharge of a watery fluid from the right nostril, possibly cerebro-spinal fluid. In this case the only cranial nerve damaged beside the fifth nerve was the third nerve; there was no evidence whatever of injury to the seventh or ninth nerves, and yet the loss of taste was obviously complete on the whole of the right side of the tongue.

In all these four cases of total fifth nerve paralysis the loss of taste on the tongue is difficult, if not impossible, to explain except by accepting a gustatory function for the fifth nerve. All four of these cases were carefully and repeatedly tested for the sense of taste, and in two of them the loss of taste was complete on the back of the tongue and palate as well as on the front of the tongue.

On the other hand, the following case is interesting for comparison.

A man, *æt.* 29, developed gummatous meningitis, with total paralysis of the right seventh and eighth nerves, with partial right fifth nerve paralysis; taste was totally lost to sweet, salt and bitter on the anterior two-thirds of the tongue, though fairly acute on the back of the tongue. Two months later the fifth nerve weakness had almost cleared up under treatment by biniodide of mercury, though the facial paralysis was still complete and the sense

of taste was still almost completely lost on the right side. In this case the nerve of Wrisberg appears to be the only gustatory nerve for the front of the tongue.

The following case of apparently uni-lateral pontine thrombosis, with total bi-lateral ageusia, or complete loss of taste, front and back, on both sides, is evidence of the probable median path of the gustatory fillet in the tegmentum.

A man, *æt.* 46, had a "stroke" one morning, without any loss of consciousness, developing suddenly loss of power and numbness over the whole of his right side. The right knee-jerk was increased and the right plantar response was extensor, and there was total right hemi-anæsthesia up to the middle line, including the face, even a hard squeeze on the arm or leg not being felt.

There was total loss of the sense of position in the right arm, which, as he said, got "lost in bed," and when lying on his right side in bed he said he felt as "though he was suspended in the air under the bedclothes."

At first there was complete bi-lateral loss of taste, though the anæsthesia included only the right half of the tongue and palate, but not the pharynx.

Taste afterwards improved on both sides, but, as the pupils at first lost their light reaction, the lesion was probably high up in the pons, in the tegmentum well above the level of the fifth nerve nuclei. The bi-lateral loss of taste on both front and back of tongue and palate is probably to be explained by the thrombotic lesion in the tegmentum, which produced the dense cutaneous anæsthesia by involving the fillet, also damaging the gustatory fillet of each side, which appears to run close to the median line in the tegmentum, on the outer side of the posterior longitudinal bundle (see Fig. 35).

Indeed, bi-lateral loss of taste, with the remaining signs strictly one-sided, except possibly bi-lateral facial anæsthesia, may be taken as evidence of a lesion on one side of the pons reaching the middle line at and above the level of the fifth nerve nucleus.

It is indeed stated that the trigemino-thalamic tract remains separate and reaches a special portion of the thalamus, and it would also appear that the gustatory fillet is also separate and runs at first close to the median line in the reticular formation, dorsal and external to the posterior longitudinal bundle.

Complete bi-lateral ageusia on the front two-thirds of the tongue only was also present in a boy of 14, whom autopsy showed to have been suffering from glioma of the left crus cerebri and upper portion of the pons on the left side. There was present right-sided spastic hemiplegia and dense hemi-anæsthesia including the right side of the face and head, while in addition there was tactile loss over the left trigeminal area, though there was no loss to pin prick or to temperature on the left side of the face. The left masseter and temporal muscles were also paralysed, though the right masseter could be felt to contract.

The dissociation of sensation on the left side of the face, and the left motor fifth palsy, indicated a lesion at the level of the left principal sensory nucleus of the fifth nerve, involving the upper halves of the gustatory nuclei on each side, but the spinal root of the left fifth nerve and the lower halves of the gustatory nuclei escaped. The boy could taste acutely on the back of his tongue on the left side, but not on the right side, either on the front or back. There was left facial paralysis, and paresis of the left sixth nucleus causing loss of the conjugate movement of the eyes to the left. There could be little doubt that the growth invading the tegmentum in the crus was the cause of the right hemi-anæsthesia and the right-sided loss of taste by involvement of the gustatory fillet, while the loss of taste on the front of the tongue on the left side was due to the growth involving the upper two-thirds of the gustatory nucleus as well as the upper sensory and the motor nuclei of the fifth nerve. Since taste was preserved on the back of the tongue on the left side the lower part of the gustatory nucleus which receives the ascending root of the glosso-pharyngeal nerve must have

escaped, and its central prolongation in the gustatory fillet of the opposite side would also escape damage.

Bi-lateral ageusia on the front two-thirds of the tongue was also present in a solicitor, *æt.* 52, whom I saw twice and examined most carefully in 1912. He was a very intelligent man, and his answers to the tests were quick and definite. He was a sufferer from chronic syringobulbia, involving many cranial nerves on the left side. There was total left fifth nerve paralysis, coarse nystagmus, partial left facial paralysis, left nerve deafness, and paralysis of the left soft palate and vocal cord, with diminished sensation on the left side of the pharynx. Taste was completely lost on both sides of the tongue as far back as the circumvallate papillae.

On the back of the tongue he could recognise at once sweet, salt and bitter, though less acutely on the left side. There was no sensation of taste at all on the soft palate on either side unless it was allowed to touch the back of the tongue, when he named the taste sensation at once. There was no detectable sensory lesion on the right side of the tongue or face to pin prick, temperature or light touch, though crossed analgesia and thermanæsthesia were present on the right side of the back of the head and neck and over large areas on the side of the trunk and right arm and lower extremity.

This case does not distinguish between the trigeminus and the nerve of Wrisberg as the gustatory nerve for the front of the tongue, but it certainly excludes the glosso-pharyngeal. The case is, however, of great interest in illustrating bi-lateral ageusia on the front of the tongue in an intra-pontine lesion, without any evidence of lesion of either the right fifth or seventh nerves. It may probably be explained by the syringal gliosis extending across the middle line and involving the upper halves of both gustatory nuclei without damaging the gustatory fillet on either side. Unlike the case of pontine thrombosis previously described, there was no evidence in this case of the lesion spreading higher than the main fifth nerve nucleus, and the

retention of acute sensation of taste on the right side of the back of the tongue negatived the possibility of explaining the loss of taste on the right front of the tongue by an extension of the gliosis on the left side forwards to involve the gustatory fillet above the level of the fifth nucleus.

This patient also illustrated another point of interest, in that for many years he had been subject to attacks of vertigo severe enough to make him stagger as if drunk, with at the same time the appearance of stinging pains in the left cheek, and flushing of the face. These symptoms may be ascribed to the lesion involving the spinal root of the fifth nerve and the inferior cerebellar peduncle.

Doyle[1] published a case in 1923 of paroxysmal neuralgia in which the sensory root of the right trigeminal nerve was avulsed, and later the glosso-pharyngeal nerve on that side also. After the second operation total loss of taste was found on the right side, both on the front and back of the tongue and palate. Taste was not tested before the second operation on the glosso-pharyngeal, but it can scarcely be suggested seriously that the glosso-pharyngeal can be the gustatory nerve for the front two-thirds of the tongue. In this case there is no reason to ascribe the loss of taste to the nerve of Wrisberg, and it seems impossible to deny the gustatory function of the fifth nerve in this case.

The most convincing evidence that I can present of the gustatory function of the fifth nerve, both of the second and third divisions, is the results of careful tests of cases of trigeminal neuralgia in which I have injected the foramen ovale with 90 per cent. alcohol, producing complete anæsthesia of the third division of the fifth nerve. In some of these cases there was also produced partial anæsthesia of the first and second divisions, what may be called partial ganglion anæsthesia, and in scores of cases permanent and total ganglion anæsthesia has resulted, in which all three divisions of the fifth nerve remained completely anæsthetic to all forms of stimuli.

Cases such as these practically amount to physiological

[1] *Arch. Neurol. and Psych.*, 1923, Chicago, Vol. IX., p. 34.

experiments, and I find it impossible to reconcile my results with those published by Cushing and Morriston Davies, whose examination of the sense of taste after Gasserian operations showed scarcely any cases with permanent loss of taste.

Analysis of my notes of cases of injection for trigeminal neuralgia shows eighty-six cases in which total loss of taste on one side of the tongue was found immediately after injection of the foramen ovale, though taste was at once recognised on the other side. In addition to these notes I have many times demonstrated uni-lateral loss of taste in hospital cases after such injection, of which the notes are incomplete. Of late years I have not tested for taste regularly, as I had found loss of taste on the one side so constantly, with few exceptions, that I considered the point as proved to my satisfaction. In only six cases can I find a record of taste being preserved, or only slightly diminished, when dense anæsthesia of one side of the tongue had been produced as the result of injection of the foramen ovale, but in addition to these I can remember having noticed the point of preservation of taste in perhaps half a dozen more hospital cases, immediately after injection of the third division. In a few other cases taste was totally abolished at first, but returned later, though the tongue remained anæsthetic.

The twenty-nine cases described at the end of this chapter include twenty-one in which loss of taste persisted on the anterior portion of the tongue, after injection of the foramen ovale, for periods of over three weeks, six of them being observed from one to eight years after the injection. In addition to these I have notes of numerous other similar cases in which loss of taste was recorded immediately following the injection, and persisted as long as the patient remained under observation, for periods varying from a few minutes to several months, e. g. case on p. 263.

Against these must be compared the few cases in which there is no loss of taste at any time, and those cases in which initial loss of taste recovered entirely, although the tongue remained completely anæsthetic.

Of these I have seen several, though they are, in my experience, far less numerous than those in whom the loss of taste is persistent. How can these apparently conflicting facts be reconciled?

One possible explanation is that in certain individuals when the usual path for taste impressions is blocked by disease or by operative procedure, an alternative route may be made use of, of which at least five are anatomically possible. Tracing them centralwards, they are :—

(1) Chorda tympani, geniculate ganglion, pars intermedia Wrisbergi.
(2) Chorda tympani, geniculate ganglion, great superficial petrosal and Vidian nerves to Meckel's ganglion and second division of the fifth nerve.
(3) Chorda tympani, geniculate ganglion, small superficial petrosal nerve to otic ganglion and third division of the fifth.
(4) Chorda tympani, superficial and deep petrosal nerves and tympanic plexus to nerve of Jacobson and glosso-pharyngeal nerve.
(5) Lingual nerve direct to third division of the fifth nerve.

The last route is probably scarcely ever employed, except in those rare cases of congenital absence of the facial nerve due to non-development of the internal and external ear and the consequent non-development of the nerve of the first gill-slit, the facial nerve.

Experience of cases of old complete facial paralysis from mastoid operation shows invariably permanent loss of taste on the front of the tongue, which may be taken as proof of the taste function of the chorda tympani.

Unless the taste fibres leave the chorda at the geniculate ganglion and pass by route (3) to the otic ganglion and the third division of the fifth nerve, how is it possible to reconcile complete loss of taste on the front of the tongue, which has been shown to occur so frequently in fifth nerve paralyses and after neurolysis by alcohol injection of the third

division at the foramen ovale, with the gustatory function of the chorda tympani?

Yet this roundabout path *via* the small superficial petrosal nerve and the otic ganglion seems too romantic to be possible, and, moreover, it is just as probable that the small superficial petrosal nerve does not conduct sensory impulses towards the otic ganglion and the fifth nerve, but from the otic ganglion towards the geniculate ganglion and the pars intermedia Wrisbergi.

If, then, the postulate be accepted that the chorda fibres are continued centralwards to the gustatory nucleus in the pons by means of the nerve of Wrisberg, which indeed Nageotte's [1] work conclusively proves, the question at once arises, What is the connection between destruction of the third division of the fifth nerve and the loss of taste which so frequently instantly follows it?

Rejecting Nageotte's suggestion as insufficiently proved, that the lingual nerve is also gustatory in function, an answer to the problem may be found if we consider that taste is a complex function, requiring in many people, probably the majority, the presence of common sensibility of the mucous membrane of the tongue and palate for the proper appreciation of gustatory stimuli, and that in these persons an interdependence of the activities of the lingual nerve and the chorda tympani is necessary for the full development of a taste sensation, and that a lesion of either path is therefore sufficient to prevent either from functioning alone as a gustatory nerve. This is probably the clue to the explanation of Nageotte's description of trigeminal fibres ending in the upper portion of the gustatory nucleus in proximity to the terminations of the pars intermedia Wrisbergi and even of the glosso-pharyngeal, rather than that we should consider that each of these nerves is self-contained in its gustatory function.

An illustration of the probability of such correlation of function in these two nerves is evident when careful tests of the sensations of taste are undertaken. If the tongue

[1] *Rev. of Neur. and Psych.* Edinb., 1906, Vol. 4, p. 473.

is kept protruded and gently dried while various tests are applied to the side of the tongue in solution, such as syrup, saline and quinine solutions, the majority of individuals are unable to indicate, by pointing to printed labels, which of these tests is being used, though evidently some gustatory sensation is perceived, and it is not until they are permitted to withdraw the tongue into the mouth and move it about that the full sense of taste is developed. Thus, in all my tests of the sense of taste, if the patient indicates by nodding the head that taste is perceived on the tongue, I accept that as a positive result, even though the quality of the taste may be uncertain until the tongue is returned within the mouth. Once the tongue is thus withdrawn within the mouth there can, of course, be no certainty that the testing material does not quickly reach the opposite side, and it is therefore advisable when attempting an accurate test to see that the tongue is kept protruded until the answer to the test is indicated.

Occasionally individuals are met with who are unable to perceive any sensation of taste when solutions are painted on the tongue so long as the tongue is kept protruded, though as soon as the tongue is withdrawn into the mouth the correct taste sensation is appreciated and named within two or three seconds. The following case is an example of this point, and also illustrates a severe form of dental neuralgia, simulating trigeminal neuralgia major.

A lady, *æt.* 35, was sent to me by a dentist on November 17th, 1923, on account of severe constant pain in the right lower jaw, starting like an ordinary toothache, and much aggravated by extraction of two teeth, one of which broke. She described the pain as unceasing, but jumping and running along to the right lower lip. She was afraid to wash or touch the face, and eating made the pain worse. The latter points were very suggestive of true neuralgia major, but it differed in that the pain was present constantly during waking hours, and was aggravated on lying down. I injected the foramen ovale under an anæsthetic on November 30th, producing total third division anæsthesia.

The pain did not disappear at once, but improved steadily from that time, longer and longer periods of complete freedom from pain occurring until the pain finally ceased four weeks after the injection. When seen again on March 25th, 1924, four months later, she was still quite free from pain, and there was still total anæsthesia of the right third division of the fifth. Testing taste with the tongue protruded, she was unable to recognise any sensation of taste on either side of the tongue to syrup or quinine solution, even after the lapse of several seconds, until the tongue was withdrawn into the mouth and moved about, when she was able to tell correctly when the left side of the tongue had been painted, but no response was obtained from the right side, and she stated that she could taste nothing whatever on the right side of the mouth.

In a few cases I have noticed that some sensation to pin prick and scratch persists on the tongue, especially towards the tip, after injection of the third division or of the whole ganglion, when all other indications point to a total destruction of the third division or of the whole ganglion. I have also noticed the same persistence of sensation on the tongue, associated with preservation of the sense of taste, after gasserectomy.

A lady, *æt.* 51, had been a sufferer from bi-lateral neuralgia major of the fifth nerve since the age of 24. The pain began suddenly on the right side, and within a year affected the left side also, rather more severely. Gasserectomy was performed on the left side in 1908, since when she had no pain on that side, though the neuralgia continued on the right side. Twelve years afterwards, I saw her in 1920 for injection of the right ganglion, and in Oct. 1925 she was still quite free from recurrence of pain. I found total anæsthesia of the left fifth nerve area, except of the tongue, where she could feel a scratch quite well, and taste sensation was quite acute.

This apparent anomaly is, I think, to be accounted for by an unusual degree of sensory supply by the chorda tympani to the side and tip of the tongue (cf. p. 321 in the section on Geniculate Neuralgia, a man who complained of numbness on the right side of the tongue and on the right lower lip, with right-sided loss of taste following

geniculate herpes and facial palsy, with numerous herpetic vesicles on the side of the tongue and on the soft palate on that side).

On two occasions I have seen persistent partial sensation on the lower lip and side of the tongue, with continuance of the neuralgia, after alcohol injection of the foramen ovale, which was unaltered by two further Gasserian operations, first, gasserectomy, and as no further benefit occurred, section and avulsion of the sensory root of the fifth nerve behind the ganglion was done as a last resort. In both the patients referred to, women of over 50, paroxysmal major neuralgia had existed for several years, and there was no reason to suppose the pain was a psychalgia.

Explanation is difficult. In each case two surgeons had done the operations, both well skilled in the Gasserian operation, so that it seems unlikely that any third division fibres could have escaped section.

I am inclined to think that the chorda tympani and the geniculate system may be in such cases an alternative path for tactile sensibility and for the painful neuralgic spasms (cf. Painful Tic Convulsif, p. 324). In a few cases I have seen after gasserectomy or after alcohol injection of the foramen ovale, persistence of partial sensation to touch and prick on the lower lip and chin, when the remainder of the third division was totally anæsthetic, including the half of the tongue, and the neuralgia had completely disappeared. In such cases, as in the cases of gasserectomy described above and on p. 276, I am inclined to think the lower lip and chin must have an alternative sensory supply from the cervical plexus.

CLINICAL CASES OF ALCOHOL INJECTION

To illustrate the gustatory function of the fifth nerve.

A lady, *æt.* 66, seen in private practice, had suffered from neuralgia of the left second and third divisions of the fifth nerve for sixteen years. On June 24th, 1913, I injected her left Gasserian ganglion through the foramen ovale by my usual method. Total ganglion anæsthesia

was produced for ten minutes, but this gradually faded partially from the first and second divisions, leaving total anæsthesia to all forms on the third division, with motor fifth nerve paresis, and complete loss of taste on the left side of the tongue to sweet, salt and bitter. Six days later there was no change, complete loss of taste on the left side still being present, but five months later, when seen on November 26th, 1913, I found slight recovery of sensation on the chin to pin prick, though no sensation to pin or scratch on the left half of the tongue. There was still medium analgesia on the second division, but little on the forehead. Taste was then found to be equally intense on the two sides of the tongue, though more slowly recognised on the left side. Six years later the late Sir John Tweedy, after examining her eyes, wrote to me that " there has been no return of neuralgia, but the left half of the face is anæsthetic up to the middle of the lip."

I find similar results now in those cases which I have recently tested; *e.g.*, a lady, *æt.* 56, had for five years been subject to paroxysmal neuralgia of the right second and third divisions. On February 15th, 1924, under local anæsthesia only, I injected the right Gasserian ganglion through the foramen ovale, 0·5 cubic centimetre of 90 per cent. alcohol causing total ganglion anæsthesia. Tests carried out immediately afterwards showed total loss of taste on the right side of the front of the tongue and palate, and on the following day she volunteered that she could taste nothing on the right side. There was no subjective consciousness of any alteration in the feeling of her face in spite of the anæsthesia being total. Slight keratitis developed on the third day, but was easily controlled by keeping the eye closed, and using boracic irrigation. Three days later when she left the nursing home there was still total ganglion anæsthesia on the right side, and the right-sided loss of taste was still complete.

Though the initial loss of taste after injection of the third division of the fifth or of the Gasserian ganglion may recover in some cases in spite of the persistence of total anæsthesia of that side of the tongue, yet in many other cases I have found the loss of taste persistent for many months and even years after injection of the nerve.

A man I saw in October 1913 had been injected by

Ostwalt in 1907, using Schlosser's intra-oral method, after which he was free from neuralgia for six years. When I saw him the tongue was still practically completely anæsthetic on the left side, with complete loss of taste, which was still unaltered two years later, or eight years after Ostwalt's injection, though pain was then recurring in the lower jaw. An interesting point in this case was the evidence of heredity of the neuralgia; he himself had suffered for twenty years, and his mother had suffered similarly for many years from the same type of tic douloureux.

The following case of bi-lateral total ganglion anæsthesia exhibited bi-lateral total loss of taste on the anterior two-thirds of the tongue. This case and the next have already been quoted in earlier chapters.

In February 1913 I injected a woman, *æt.* 41, at the National Hospital for the late Dr. Batten; she had had severe neuralgia of the right second and third divisions for eight years. Under chloroform I injected the right foramen ovale with 1·75 cubic centimetres of 80 per cent. alcohol, the foramen being found without any difficulty. As a result, total right ganglion anæsthesia was produced, with complete loss of taste on the right front of the tongue, both of which conditions have persisted unaltered ever since without any return of the neuralgia. Five years later similar neuralgia commenced on the left side of the face, and in December 1919 I injected the left foramen ovale under local novocain only instead of chloroform, injecting two cubic centimetres of 90 per cent. alcohol into the ganglion. Total left ganglion anæsthesia resulted, with loss of taste on the left front of the tongue, and weakness of the left masseter, though the right masseter had long recovered completely. When examined seven weeks later there was still total anæsthesia of both sides of the face, forehead, eyes, tongue and jaws, with total loss of taste on both sides of the tongue in front of the circumvallate papillæ. As she said, "All food feels like chaff" in the mouth until she swallows, when she can taste it perfectly.

This case is equal to the most perfect physiological experiment in destroying both Gasserian ganglia without producing any lesion of the seventh or ninth nerves, and the resulting total and persistent loss of taste on the front

of the tongue is conclusive evidence of the important gustatory function of the fifth nerve, the loss of taste on the right side having been observed for nearly seven years after injection of the right ganglion.

A man, *æt.* 59, began to suffer from neuralgia, in some respects resembling neuralgia major of the second division, round the left eye and side of the nose, and in the left upper gum, complaining of "jumping pain, quick as a flash of lightning, like a flaring fire." The neuralgic attacks were usually worse in the cold weather, but were not aggravated by eating, talking, or rubbing the face. When I saw him, six years after his pain commenced, there was no trace of anæsthesia, but the severity of the pain was increasing, and alcohol injection of the supra-orbital nerve and of the second division at the foramen rotundum gave only partial relief, though deep anæsthesia was produced. His taste at this time was tested and was found equally acute on both sides of the tongue. A fortnight later I injected the left foramen ovale, producing total ganglion anæsthesia for a few minutes, which then wore off the first and second divisions partially, leaving total anæsthesia of the third division. The pain still continued round the eye for a fortnight, and then diminished very much, so that he might have no neuralgia at all for two or three weeks together and then have slight attacks for a few minutes only. When I saw him eighteen days after the injection of the foramen ovale he complained that he could not taste at all on the left side of his mouth, and on testing I found that he had no sense of taste whatever on the left front half of the tongue, using salt solution and syrup, though both of these were at once recognised on the right side of the tongue. Four and a half years later there was still complete absence of taste on the left half of his tongue, which was still anæsthetic, though sensibility and neuralgic pain had returned over the second division. Nine months later, in June 1919, his neuralgia was again becoming severe, and I therefore injected the ganglion through the foramen ovale, on this occasion destroying it completely, producing total and permanent anæsthesia of the whole fifth nerve area on the left side. His doctor wrote in February 1924, four and a half years after the ganglion was finally destroyed by injection, that he still had complete loss of taste on the left half of the tongue, and was quite well, except for occasional shooting pains on the left side of the face, his age

then being 75. Although this case in some respects differed from ordinary chronic paroxysmal trigeminal neuralgia, yet it is none the less instructive in demonstrating the gustatory function of the fifth nerve.

A lady, *æt.* 63, had suffered for sixteen years from neuralgic pains in the left lower jaw, later also affecting the second division. The neuralgia was commonly started in the usual way by eating, talking or washing the face.

On July 19th, 1913, I injected the left foramen ovale, producing complete anæsthesia of the lower lip and tongue, but less complete of the auriculo-temporal area, and I also injected the infra-orbital nerve, producing anæsthesia of the upper lip and cheek. When examined ten months later, and again two and a quarter years after the injection, there was still complete anæsthesia on the left side of the tongue, with total loss of taste on the left side of the front of the tongue, though taste was quite acute on the right side.

A lady, *æt.* 44, unmarried, when first I saw her in July 1912, had been suffering severely at intervals for eighteen months from neuralgia major of the right third division. At first the agony in the lower jaw was very intense, " she thought such pain was impossible." The acute attacks lasted about a week, she could neither eat nor drink, the pain scarcely ever ceasing, and stinging into the right side of her tongue. She called it a " hellish pain," and during the attacks said she felt as though she could smash everything, or commit suicide. I injected her right foramen ovale, under morphine and hyoscine, producing deep anæsthesia of the lower lip, tongue and chin, with partial analgesia of the auriculo-temporal area. There was immediate loss of taste on the right half of the tongue, syrup only being used, but this was instantly recognised on the left side of the tongue. When seen again seventeen months later, pain was returning in the lower jaw and lip, but the right side of the tongue was still almost completely anæsthetic, and taste was still completely absent on the right side of the tongue, though sweet and salt were recognised at once on the left side.

Another lady, *æt.* 71, I saw in June 1917 for chronic trigeminal neuralgia of the right side, from which she had suffered for no less than fifty-four years, the pain having begun in paroxysms on the right side of her face at the age

of seventeen. This had continued at intervals ever since, gradually becoming much more severe during the last ten years. On June 3rd, 1917, under gas and oxygen, I injected the right Gasserian ganglion through the foramen ovale, producing total anæsthesia of the whole fifth nerve. Fourteen months later there was still practically total ganglion anæsthesia, with the exception of slight sensibility on the right upper lip, and there was complete loss of taste on the right half of her tongue, taste being normally acute on the left side.

A young woman, *æt.* 32, in January 1911 was exposed to a very cold wind when motoring for five miles over the moors in the North of England after travelling in a heated train. Eight hours later she was struck by sudden severe pain in the left cheek, like red-hot wires jumping, in spasms every minute or two if she moved or talked, or when eating. These paroxysms continued with varying intensity until November 1912, when I injected the left foramen ovale, producing at first total ganglion anæsthesia, which passed off the first and second divisions after ten minutes, leaving complete anæsthesia of the third division, with paresis of the left motor root and loss of taste on the left half of the tongue, which persisted unaltered six and a half months later, taste being quite acute on the right side.

A woman, *æt.* 49, had been suffering from paroxysmal neuralgia of the left second and third divisions of the fifth nerve for five years. The pain had gradually been increasing in intensity so that she could scarcely wash her face, and eating and drinking also started the pain. I first saw her on June 27th, 1919, and on the same day I injected the left Gasserian ganglion, causing total anæsthesia of the whole of the left fifth nerve. Twelve days later there was still total ganglion anæsthesia, both to pin and pressure, the anæsthesia including the whole of the soft palate, and there was total loss of taste on the left half of the tongue. The eye gave no trouble, and four months later there was still total trigeminal anæsthesia and motor paresis with complete loss of taste on the left half of the tongue. She said the face felt uncomfortable, as though the skin were drawn tight, but not a swollen feeling. A year later she was still quite well, no recurrence of pain, still totally anæsthetic, but with no subjective sensation of discomfort; taste was not tested on that occasion.

A woman, *æt.* 52, when seen in April 1913, had had spasmodic pains in the right lower jaw for five years. Latterly the pain had been spreading into the lip and tongue so severely that for two nights she was almost crazy. There were trigger zones on the lip and cheek, and eating or talking would start the pain. Injection of the foramen ovale on April 23rd, 1913, under morphine and hyoscine, produced complete anæsthesia of the whole of the right third division, with loss of taste of the right anterior portion of the tongue, taste being acute on the left side. Three months later there was still complete anæsthesia of the chin, lip, lower jaw and half the tongue, though sensation was commencing to return over the auriculo-temporal area. There was practically no perception of taste on the right half of the tongue, though the tests were immediately recognised on the left side.

A woman, *æt.* 31, a case of bi-lateral trigeminal neuralgia, had been suffering ever since the age of sixteen from shooting pains in the left third division and sometimes in the right second division, with only occasional intervals of freedom; she said the pains were like electric wires, and all the teeth had been extracted without benefit. When I saw her in February 1920 the painful spasms had been almost continuous for six months, and any movement of the jaw or sudden noise would start the pain.

Injection of the left foramen ovale on February 28th produced total anæsthesia of the third division, with temporary loss of taste, but the anæsthesia practically disappeared after an hour and the neuralgia returned.

This was doubtless due to novocain, not alcohol anæsthesia, and no alcohol could have been injected into the nerve itself. Three days later, I injected the left foramen ovale again, and got total third division anæsthesia; then, moving my needle slightly further back and pressing it another quarter of an inch deeper, injection of 0·5 c.c. more alcohol produced total ganglion anæsthesia with motor palsy and loss of taste on that side. The eye was kept closed for some days, but gave no trouble. On June 1st, 1920, three months later, the conditions were unaltered, no perception of taste to salt and quinine on the left front of the tongue. On August 23rd, 1921, seventeen months after the injection, she still had total left ganglion anæsthesia, except of the eyeball, on which pressure was then perceived, and the left masseter had partly recovered.

There was no recurrence of neuralgia on the left side, but similar neuralgia was then affecting the right second division, which was accordingly injected at the foramen rotundum, causing complete anæsthesia of its distribution, including pressure, the anæsthesia being complete on the hard palate and the anterior two-thirds of the soft palate, nearly to the base of the uvula.

I saw her again, August 14th, 1923, three and a half years after the first injection, when I found total anæsthesia of the left second and third divisions still persisting, with complete analgesia of the first division. She had had no recurrence of pain on the left side, and there was still a good deal of numbness of the right second division, with no recurrence of neuralgia since she was injected two years previously, though latterly she had been having neuralgic spasms in the right third division.

She said that ever since the pain originally started at the age of sixteen, the pain on the right side of the face used to alternate with that on the left side, though the left side was always the worst. I have no note of any examination of the taste sensation since three months after the injection of the foramen ovale, when the loss was still complete. She said she did not notice the numbness on the left side of her face at all.

The wife of a bank manager, *æt.* 41, was seen for severe left-sided trigeminal neuralgia in October 1911, which had started five years previously like a toothache, five teeth being removed without benefit. This was followed shortly afterwards by extremely severe neuralgia, for ten minutes at a time, recurring every hour or two, some of the attacks lasting as long as twenty minutes. She compared the pain to red-hot corkscrews being plunged into her face, the original severe pain having felt as though thousands of needles had been flung at her face. Two years before I saw her she had had a series of injections of alcohol under general anæsthesia.

This relieved the pain, but caused troublesome diplopia for three months. When I saw her for recurrence of the neuralgia the diplopia had long disappeared, but the left pupil was typically Argyll Robertson in character, being medium small and quite fixed to light, though contracting on convergence. This was the only permanent sequel from the partial third nerve injury following the injections. On October 16th I injected her Gasserian ganglion, producing analgesia over the first division of the left fifth nerve, and

total anæsthesia to all forms on the second and third divisions, except for pressure on the lower lip and chin, and taste was completely lost on the left side of the tongue. Most of this anæsthesia had disappeared within three or four days and taste was recovered on the left side of the tongue.

Ten days after the first injection I again injected the ganglion, using two cubic centimetres of spirit, again producing total fifth nerve sensory loss, including loss of taste. After this injection the anæsthesia scarcely altered, remaining total on the first division, and slight keratitis developed, which was cured by fastening the lids together for a month.

Scarcely any sensation returned on the second division, but pricks were just perceptible on the left side of the chin and lower lip. Taste remained completely lost on the left front two-thirds of the tongue, though it was quite normal on the back of the tongue and on the posterior border of the soft palate. Taste was most carefully tested on several occasions, with the above unvarying result, the last occasion being on January 10th, 1912, eleven weeks after the second injection of the ganglion.

My first Gasserian ganglion alcohol injection[1] I performed on November 28th, 1910, for a man, *æt.* 43, with chronic trigeminal neuralgia. Immediately after the injection there was total fifth nerve anæsthesia, though on the next day the sense of pressure and moderate tactile sensibility had returned over the first and second divisions, but there was still complete right third division anæsthesia, with total loss of taste on the right front of the tongue. Four days later pressure sense returned on the right side of the tongue and chin, and taste returned partially on the right half of the tongue, though less acutely than on the left side. When examined thirteen months later there was still local analgesia and loss of light touch sensation over the whole right fifth nerve area, and he complained that he did not taste his food so well on the right side as on the left, especially sweet substances.

On testing the sensation of taste it was found to be obviously diminished on the right side, recognition especially being much slower than on the left side. When last I heard of him in 1918, seven and a half years after the ganglion injection, he was still quite free from the neuralgia.

[1] *Lancet*, January 27th, 1912, p. 218.

On October 22nd, 1911, I injected the left third division at the foramen ovale in an unmarried lady of 47 for trigeminal neuralgia, causing total anæsthesia of the inferior dental and lingual areas, and partial anæsthesia of the auriculo-temporal. There was immediate total loss of taste on the left half of the tongue to salt and sugar, both of which were almost instantly recognised on the right side. Nineteen days later there was still total loss to salt and sweet on the whole of the left half of the tongue, and the hard and soft palate on that side. She complained, moreover, that she could taste nothing at all on the left side.

On July 5th, 1911, I injected the right foramen ovale of an unmarried lady, *æt.* 35, for chronic neuralgia of the right lower jaw which she had suffered from for the previous four years. There was immediate anæsthesia produced of the branches of the third division, though not total, pin pricks on the lower lip being felt as touches. Sugar was not tasted on the right half of the tongue, but was recognised at once on the left side. Next day the anæsthesia and loss of taste remained unaltered on the right side, though she could recognise taste sensations on the back of the right side of the tongue.

There was well-marked anæsthesia to touch and pin on the front of the ear, on the tragus down to the incisura notch, and inside the external auditory meatus, along its anterior and upper wall, down to the membrane, which was also comparatively anæsthetic. The floor of the meatus was less anæsthetic, and pin prick was felt sharply on the posterior and upper wall. There was no anæsthesia of the concha; indeed on only a few occasions have I seen this portion of the auricle anæsthetic after trigeminal injection.

On July 26th, three weeks after the injection of the foramen ovale, the anæsthesia of the third division was still just as complete, but slight taste sensation had now returned on the right half of the tongue. When a lump of sugar was rubbed on that side of the tongue, no taste sensation was perceptible for several seconds, and even then it was very faint, though she instantly recognised sugar when the piece was rubbed on the left half of the tongue.

On December 4th, 1913, I injected the left foramen ovale in a man, *æt.* 56, who had had trigeminal neuralgia of the left third division for eight years. Complete anæsthesia

over the whole of the third division was produced. Fifteen days later the left tongue and jaw were totally anæsthetic, and he complained of losing his food on the left side, and that he could taste nothing on that side. When tested with salt, there was no sensation of taste on the left side, though saline was recognised at once on the right side of the tongue. When last seen on April 11th, 1914, or over four months since the injection, there was still total anæsthesia of the left third division, including well-marked anæsthesia to pin prick on the anterior wall of the external auditory meatus and the tympanum. Taste was still completely lost on the left half of the tongue, though it was acute on the right side. There was still marked motor weakness of the masseter, though the temporal muscle showed signs of improvement.

On July 11th, 1913, I injected the Gasserian ganglion of a carpenter, *æt.* 41, for neuralgia of nine years duration in the right second and third divisions, causing total anæsthesia of the whole of the fifth nerve area, with motor palsy and loss to deep pressure on the eyeball, forehead and face, with complete loss of taste on the right anterior two-thirds of the tongue. By the following day the area of anæsthesia had shrunk somewhat, the line of anæsthesia now not including any part of the ear, but passing about an inch in front. In other respects the anæsthesia was just as complete, and indeed was still unaltered fifteen months later, on October 1st, 1914. The day after the injection the cornea became steamy, and uveal congestion was noticed, so that the eyelids were closed for some weeks. The cornea afterwards remained quite healthy. Taste remained completely lost on the right front of the tongue, but syrup was recognised at once when placed on the back of the tongue on the right side, and taste was acutely perceived also on the left half of the tongue. When I saw the man fifteen months later I made a note that he was still completely anæsthetic on all three divisions, no return of pain, and cornea quite healthy, but I have no note of taste having been tested on that occasion.

On July 13th, 1923, I injected the Gasserian ganglion of an old woman of 72 for neuralgia of thirty years' duration. The anæsthesia of the whole fifth area was total, and remained unaltered when seen in February 1924, ten months later, though the lids had been sewn up for a time

on account of keratitis. On the day following the injection she volunteered that she could not taste on the left side, and this was found to be so on testing, though ten months later taste had returned on the left half of the tongue and was practically equal on the two sides.

On March 17th, 1913, I injected the right third division at the foramen ovale in an old gentleman, *æt.* 73, causing complete anæsthesia, including pressure sense on the lower lip and chin and half of tongue, with less intense anæsthesia of the auriculo-temporal and buccal branches. There was immediate total loss of taste on the right front half of the tongue, salt and sweet only being used, though these were recognised at once on the left side. Ten days later the anæsthesia and the loss of taste on the right half of the tongue remained unaltered, the neuralgia having disappeared.

In February, 1911, I saw a lady, *æt.* 57, who had suffered from neuralgia for thirteen years, for which the usual remedy of removing all the teeth had been tried, but with the effect of aggravating the pain. In 1908 she had the inferior dental nerve divided, and a year later the same surgeon operated to excise the Gasserian ganglion, but she was free from pain only for a year, and there was very little anæsthesia remaining when I saw her in 1911. Owing to the two operations she had had I found it impossible to manipulate the needle in the zygomatic fossa, owing to dense scar tissue, and I sent her to Sir Victor Horsley for a second Gasserian operation.

The ganglion was found to be quite undamaged, and was removed intact apparently, and the sensory root was also divided; fourteen days later she was very well, with no pain, with complete loss of sensation to pressure, pin prick and touch over the forehead, eye, cheek and tongue and lower lip, *but considerable sensation remained on the chin.* There was practically complete loss of taste on the right front of the tongue to a careful test, using quinine and sugar, which were both recognised at once on the left side. There was no subjective sensation on the anæsthetic side of the face. This is the only gasserectomy case that I have fully examined of late years, but its results for taste and anæsthesia appear to me indistinguishable from many of my ganglion injections (see p. 265).

On July 22nd, 1913, I injected the left foramen ovale

in a lady of 58 for characteristic neuralgia major of the inferior dental nerve. Total third division anæsthesia was produced, and moderately deep anæsthesia of the first and second divisions. On the following day the anæsthesia had almost completely worn off the first and second divisions, but remained complete over the third division, including the anterior wall of the auditory meatus and the auriculo-temporal area, with motor palsy and loss of taste on the left half of the tongue. A week later, on July 30th, 1913, there was still total third division anæsthesia and loss of taste on the left half of the tongue, with almost perfect sensation over the first and second divisions. She had had no more neuralgia since the injection, and she shortly after left for South Africa. In February 1925 I heard she was still quite free of pain, eleven and a half years after the injection.

On December 23rd, 1914, I injected the right Gasserian ganglion in a lady, *æt.* 72, who had suffered for twelve years from trigeminal neuralgia of the first and second divisions. Total fifth nerve anæsthesia at once resulted, nearly two cubic centimetres of alcohol being used. Towards the end of the injection vertigo and nystagmus appeared, the eye movement being chiefly towards the left side, and complete right facial paralysis developed. Within a quarter of an hour both the facial paralysis and the nystagmus had completely disappeared, though slight vertigo persisted for an hour or so, but there was no nerve deafness at any time. After twenty minutes the anæsthesia largely passed off from the first and second divisions, leaving total third division anæsthesia, with complete loss of taste on the right half of the tongue in front. Eight days later, on December 31st, she was quite free from neuralgia. Anæsthesia was still almost complete on the right third division, and light pin pricks were not felt on the first and second divisions. Taste was still lost on the right side, neither salt nor sweet being recognised, though on the left side they were recognised at once. There had been no reappearance of nystagmus or facial weakness.

On July 24th, 1913, I injected the left third division in a man, *æt.* 44, who had complained of stinging pain in front of the left ear in short spasms for the previous four years. The pain was not brought on by eating or talking, nor could he bring it on by any movements. He had intervals of

complete freedom from pain, but in several respects the character of the neuralgia was unlike trigeminal neuralgia major. Injection of the foramen ovale produced complete third division anæsthesia, with motor palsy and loss of taste on the left half of the tongue, which was still complete a week later. The injection was quite successful in arresting his neuralgia, as he had complete freedom for three and a half years, and then occasional twinges only for another two years, when it began to be severe again.

When seen in February, 1919, there was still slight anæsthesia of the left lower lip and tongue, and the masseter had completely recovered, but re-injection of the nerve again arrested his pain.

On October 25th, 1923, I injected the Gasserian ganglion of a woman, *æt.* 64, who had suffered from typical paroxysmal neuralgia of the right third division following the extraction of a tooth eleven months previously.

In addition to the neuralgia she had also complained of a sensation of swelling in the right side of the tongue. When seen four and a half months later, she stated that she had had no pain at all since the injection, but that the sensation of swelling in the tongue persisted and she complained that she could taste nothing on the right side. There was total anæsthesia of the whole of the right first and third divisions of the fifth nerve area, but partial sensation had returned over the area of the second division. There was total loss of taste on the right side of the tongue except at the back, where taste appeared normal. Taste sensation was quite acute on the left half of the tongue. No trouble with the cornea had been experienced.

In May 1913 I saw a lady, *æt.* 68, for severe pains around the right eye and side of the nose, who had been treated as a case of neuralgia major. The pain was not, however, characteristic in type, but all other treatment failing to relieve, I injected her Gasserian ganglion a month later, producing total ganglion anæsthesia temporarily, which after ten minutes faded off the first and second divisions partially, leaving loss to light pin prick, but total anæsthesia of the third division, with loss of taste on the tongue, which persisted unaltered three and a half months later, though sensation over the upper part of the face was by that time practically normal again.

In November, 1920, I saw a lady, *æt.* 36, who for six years

had been subject to paroxysmal neuralgia major on the left side, mostly of the second division. The pain would shoot in spasms, like " red-hot wires " in the cheek, often working up to an explosion of pain like fireworks. The pain would spread round the eye into the temple and into the nose, making her sneeze. Eating, talking, or any movements of the face would start the neuralgia, though cold did not affect it, and she was always free at night.

In 1919 and 1920 she had had several alcohol injections elsewhere without relief. On November 15th I injected both the second and third divisions through the same puncture above the sigmoid notch, producing total anæsthesia of both nerve areas. For over three years she had complete relief from pain, which recurred in January 1924, the neuralgia soon becoming severe. On February 8th I injected the foramen ovale, and attempted to destroy the ganglion, because, seeing that the patient was under the age of 40, I thought this better than recurrent injections every few years. Total anæsthesia of the first and third divisions was produced, and nearly complete anæsthesia of the second division, though pressure was perceived on the cheek. I have no note of taste having been tested on the first occasion in 1920, but after the recent injection I tested the gustatory sense at once by painting solution of quinine on the side of the tongue. There was total loss of taste on the left side, though this sense was quite acute on the right side. On March 18th, five and a half weeks afterwards, she wrote " still quite free from pain, the sense of taste has not returned to the left side of the tongue yet."

The following case in a woman of 45 is an example of a chronic neuralgia of the face differing entirely in character from neuralgia major of the fifth nerve in the constancy of the pain and in the absence of paroxysms started by eating, talking or handling the face. The pain too was worse when she was worried, and also at night, and in all probability was a psychalgia, but is included here on account of the complete uni-lateral taste loss resulting from Gasserian injection.

A lady, *æt.* 45, had been subject to constant pain in the right cheek for ten years, the neuralgia never leaving her entirely, but getting worse at night and if she

was worried. The pain began like a toothache, but was never darting or shooting in character. Most of her teeth were extracted, and the turbinal bones had been removed without improvement.

X-rays disclosed no bone or sinus disease or buried teeth, and she had consulted numerous doctors without avail. She was sent to me in May 1920, with a view to alcohol injection of the ganglion, which was done, total ganglion anæsthesia being produced, which was still complete when I saw her three months afterwards. She then volunteered that she could taste nothing on the right side, and on testing sensation, the right half of the tongue was found to be quite insensitive to taste, though the left half was normally acute. Although the pain in the face was temporarily improved, it recurred in an aggravated form some months later, and has continued in spite of all treatment, including gasserectomy.

A man, *æt.* 47, stoker in a gas-works, in July 1922 had two molar teeth extracted from the left upper jaw. He says the jaw was numbed after the extraction of the teeth, and that pain gradually began in the left upper jaw, getting worse every day. When I saw him in February 1923 he referred his pain to the left cheek only, constant and severe, almost preventing him from doing his work. There was no sign of pharyngeal growth, and though the blood Wassermann was negative he was given two intravenous injections of novarsenobillon.

The pain, however, continued unaltered, and in July he was sent up to me at St. Mary's Hospital, where I gave him two injections into the foramen ovale and to the second division at the foramen rotundum. Total anæsthesia of these nerve areas was produced, but the pain continued unabated, in fact after each injection he stated that the pain was worse than before, and it then invaded the lower jaw. He was sent home unimproved, but again returned to hospital in January 1924, for consideration of further injection for destruction of the ganglion, or for a Gasserian operation.

The areas of both the second and third divisions of the fifth nerve were still completely anæsthetic, but the man stated that the pain was constant, never leaving him for a second, and that it occupied a wider area than before I first saw him, now involving the lower as well as the upper jaw.

Further study of the case convinced me that the pain was really a psychalgia, as total anæsthesia of the second and third divisions not only did not improve the pain, but apparently made it worse. Psycho-therapy and suggestion treatment failed to make any impression, and he was sent home. The injections, I fear, did harm in fixing the pain.

When last examined in March 1924, nine months after the injections had been done, there was still practically total anæsthesia of both the left second and third division areas, with complete loss of sense of taste on the left side of the tongue, though taste was normally acute on the right side.

CHAPTER XIII

HERPES ZOSTER, TRIGEMINAL AND SPINAL

Herpes frontalis, herpes ophthalmicus, or zoster affecting the first division of the fifth nerve, is by far the commonest clinical manifestation of the trigeminal form of the disease.

Ushered in by severe pain in the forehead and eyebrow, the agony is sometimes so intense as to prevent sleep for days together. Swelling and redness of the forehead soon appear, with vesicles somewhat resembling those of modified small-pox. Sometimes the vesicles are so numerous and closely set as to become practically confluent, and raw areas of ulceration may appear, with considerable œdema of the eyelids, so that the eye may be completely closed. The herpetic vesicles may sometimes be traced down the side of the ridge of the nose, and even appear upon the conjunctiva, causing great chemosis, with keratitis of the deep layers of the cornea, perhaps followed by ulceration and scarring of the cornea, or in the worst cases by infection of the eyeball, panophthalmitis and destruction of the eye, necessitating enucleation.

In an average severe case of herpes frontalis the vesicles will be found extending backwards from the forehead into the hairy scalp as far as the vertex, and inwards to the middle line of the forehead and scalp.

With the appearance of the blebs and vesicles in the hair over the supra-orbital distribution it is not uncommon for the hair to fall off in patches, especially in a broad band along the line of the supra-orbital nerve immediately above the supra-orbital notch and one inch from the middle line (see Figs. 36 and 39).

Uveal inflammation within the eye may result from herpes of the ophthalmic division, and fixation of the pupil both to light and accommodation, with some irregularity in the shape of the pupil, is an occasional sequel.

After a few days of severe constant pain, which it is almost impossible to relieve without morphine, the intensity of the pain gradually lessens until usually after three or four weeks it amounts to little more than discomfort, with a "drawn," contracted feeling. Often paræsthesiæ of crawling and itching sensations persist for

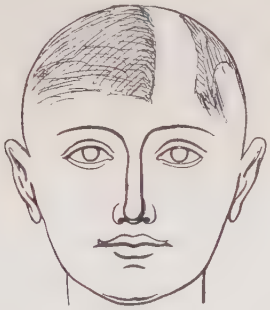

Fig. 39.—Case of Morphœa, illustrating the trophic loss of hair and atrophy of skin resulting from the trigeminal neuritis (see pp. 242–3).

many months, and in persons over 50 years of age the abnormal sensations may remain indefinitely, the liability to constant irritation and even the continuance of acute pain increasing with the age of the person attacked, so that a *chronic post-herpetic neuralgia* is not infrequent in sufferers from trigeminal zoster of over 60 years of age. I have seen severe hyperæsthesia of the whole cutaneous area of the first division of the fifth nerve still persistent thirty-five years after an attack of frontal herpes.

The etiology of herpes zoster, whether affecting the fifth nerve or one of the posterior roots, is uncertain. Many points are suggestive of the lesion being due to an infective process, and at times the cases appear to be unduly frequent, suggestive of an epidemic origin, some-

times accompanying other epidemics such as poliomyelitis. Numbers of cases of zoster have been observed to alternate with varicella, and it would seem as though there must be a very close association between these two diseases, as in numbers of instances zoster has developed after exposure to the contagion of varicella, or chicken-pox may develop in the contacts of a case of ordinary shingles whether of the face, limb or trunk. Bokay[1] first drew attention to the possible association of these two diseases. I have twice seen severe zoster affect the second division and the third division respectively in clergymen over the age of 75, in both men the shingles appearing about a fortnight after exposure to contagion from chicken-pox in a grandson. In each case the daughter who nursed the old gentleman developed chicken-pox about a fortnight after commencing duty. In a few published cases, moreover, of ordinary zona or shingles affecting the trunk the rash has afterwards become generalised, resembling chicken-pox, though the ordinary rash and vesicles of zoster do not resemble the vesicles of varicella, but appear more deeply placed and leave permanent scarring. Irritation of a spinal nerve in the neighbourhood of the posterior root ganglion may give rise to herpes along the root distribution on the skin, as in cases of spinal caries, and in new growths invading the meninges or the vertebral column. Alcohol injection of the Gasserian ganglion may in like manner cause slight herpetic eruption on the lips and chin. Zoster may also be due to prolonged arsenical treatment.

Post-herpetic Neuralgia.

After the severity of the pain of the initial attack has passed off there may be an interval in which there is considerable discomfort and a drawn sensation of tightness over the area of the rash with perhaps crawling sensations and similar paræsthesiæ. While shooting pains may be noticed, these symptoms gradually become more and more severe, so that the patient is never free from pain. This type of neuralgia, though no doubt less severe

[1] *Wien. Klin. Woch.* 1909, p. 1323.

than bad cases of paroxysmal trigeminal neuralgia, is possibly more depressing to the patient and saps his vitality and desire to live even more than the paroxysmal tic. This is probably because of the constant character of the pain, with no intervals of freedom. In a few cases movements of the face such as talking and eating may aggravate the spasms in post-herpetic neuralgia.

The frontal area or ophthalmic division is the most frequently affected of the three trigeminal branches, but the second and third divisions are sometimes badly involved, and occasionally the whole trigeminal area suffers alike. Occasionally the disease has been mistaken for erysipelas, although the most casual examination ought to distinguish this disease from zoster.

Anæsthesia is practically always demonstrable on the depressed scars following herpes, though the area may otherwise be normally sensitive, sometimes very hypersensitive. In other cases when the attack has been severe and the scarring is widespread, there may be complete loss of tactile and painful perceptions, and even loss of sensation to pressure in some cases of old frontal herpes over the whole of the supra-orbital and supra-trochlear nerve areas, though sensation is usually preserved on the eyeball and upper lid.

I have seen typical *migrainous neuralgia* as a sequel of frontal herpes. A lady, *æt.* 37, developed left frontal herpes, followed by considerable scarring over the left side of the forehead and partial loss to touch and pin prick over this area, and ever since the attack of shingles she has been liable to intermittent attacks of neuralgia in the left side of the forehead and temple, the duration lasting from half an hour to as long as two days, vomiting occurring in the bad attacks, and nausea even with the slight attacks. She had never previously suffered from headaches. In spite of the partial anæsthesia of the forehead she was extremely hypersensitive on the outer side of the forehead and temple to rubbing or to a comb or hair brush, especially during a neuralgic attack. In

view of this painful hyperæsthesia and the frequent distressing attacks of migrainous neuralgia and vomiting resulting from the herpetic lesions on the ophthalmic division, I decided to inject the Gasserian ganglion through the foramen ovale, and to produce total destruction of its inner portion, while leaving the outer portion and the mandibular nerve undamaged, if possible. This I did in July, 1925, producing total anæsthesia of the whole of the first and second divisions, while the third division recovered entirely after the novocain anæsthesia had worn off. The result was excellent, all her pain in the side of the head and the hypersensitiveness having disappeared, with no recurrence of the migrainous neuralgia three months afterwards.

In old people the after pains following shingles, whether trigeminal or of the trunk or limbs, are often compared by them to the sensation of a raw open wound. An old lady of 74, who had had zoster of the right second and third dorsal root areas twelve months previously, told me once that unless she had seen in a mirror that it was not so, she would not be able to believe that the skin was not raw, and the clothes sticking to it.

The treatment of post-herpetic neuralgia is most disappointing and difficult, and drugs such as aspirin, medinal, pyramidon and antipyrin may be tried in turn without much benefit. Analgesic liniments such as chloral-camphor and menthol may be painted on and give some temporary relief, but menthol in its various preparations should never be rubbed upon the cheek or side of the nose on account of the irritation to the eye which it produces, though for post-herpetic neuralgia of the limbs or the trunk those preparations containing menthol, chloral hydrate, camphor and methyl salicylate may be quite useful, as is also the proprietary remedy known as Frozoclone, containing menthol and Eau de Cologne.

The after pains in post-herpetic neuralgia no doubt depend to a certain extent upon the severity of the scarring and the treatment which is applied to them. Although

the actual lesion in herpes is probably hæmorrhagic, and an infective process in the root ganglia, yet undoubtedly the pains may be much aggravated and prolonged by injudicious treatment of the herpetic vesicles, which are trophic cutaneous expressions of the ganglionic lesion. For instance, I have seen the pains very much aggravated by the local injection of alcohol subcutaneously above the chief area of vesicles in the arm, in the vain hope of producing nerve block and arresting the pain. Peripheral injection of alcohol is worse than useless in cases of herpes zoster. The vesicles should be covered from the light, which is easy in the case of herpes of the trunk or limbs, but in the treatment of facial herpes the patient should either be kept in a dark room or the window covered with a red or orange blind in order to exclude the actinic rays in the sunlight. Each vesicle, moreover, should be painted separately with new skin, a non-contractile collodion, and the area must not be encouraged to perspire by the use of thick coverings. On no account should the blebs be punctured and their contents emptied. In some cases suppuration of the skin is impossible to prevent, which must then be treated on ordinary surgical principles.

Sometimes post-herpetic neuralgia of the trunk is so persistent and implacable that patients will welcome relief at practically any price, and indeed suicide has in many cases ended their sufferings.

Sir William Jenner used to describe the case of an old man who suffered from post-herpetic neuralgia before the days of anæsthetics. He endured excision of the skin of the painful area in the hope of relief from his intolerable agony, but finding none, he shot himself.

In such severe cases as these the only chance of relief is to be found in laminectomy and section of the posterior roots involved between the root ganglia and the spinal cord, or else by the more delicate operation of chordotomy.

The former operation of posterior root section is the easier of the two operations to perform, though it may be difficult during the operation to locate the posterior

roots which are required to be divided, and it is usually wiser to divide two or three more than those which appear to be involved in the herpetic eruption, in order to make sure of including all those from the damaged ganglia. Even after an operation of resection of several posterior roots the pain may persist apparently uninfluenced, and no guarantee can therefore be given of a cure by this means.

There remains the operation of chordotomy.

Chordotomy.

This is an operation devised by Spiller and performed by Frazier of Philadelphia for the relief of intractable pain such as may be produced by carcinoma in the pelvis or from any painful disease affecting the lower extremities or lower part of the trunk which cannot be relieved by other means. The operation is based on the physiological fact that the nerve fibres carrying painful sensations, after reaching the spinal cord by the posterior roots and passing through nerve cells in the posterior horn, soon decussate in the anterior white commissure and then turn upwards in the ascending antero-lateral tract on the opposite side of the spinal cord to that on which they entered it, and run upwards to join the fillet in the medulla and pons on their way to the thalamus. This ascending antero-lateral tract, as its name implies, is a bundle of nerve fibres near the periphery of the cord towards its anterior aspect, and if therefore the cord be transfixed by a fine knife across its antero-lateral margin the straight line formed by the knife will be the chord while the outer curve of the spinal cord forms the arc, or portion of a circle or ellipse.

After transfixing the cord just internal to the column of fibres forming the ascending antero-lateral tract, the edge of the knife is turned outwards and a cut is then made through the tract. In order to perform this operation, therefore, a laminectomy is done at least several segments above the highest spinal nerve conveying the

painful sensations, preferably in the upper dorsal or lower cervical region, and after opening the dura and exposing the spinal cord the latter has to be twisted round after dividing the ligamentum denticulatum, and the cord is then transfixed antero-laterally and the tract divided. When the pain is uni-lateral, as in post-herpetic neuralgia, it must be the antero-lateral tract of the opposite side that is divided by this operation of chordotomy, though when the pain is bi-lateral, as in carcinoma in the pelvis or lower vertebræ, chordotomy must be performed on each side. Several very successful results have been recorded in the relief of pain from carcinoma, and the same may be expected in post-herpetic neuralgia when the pain is entirely ganglionic in origin. It is probable, however, that in some cases of after pains from herpes, just as in cases of causalgia from damaged nerve trunks in the limbs, a mental element is introduced after a time, which may be quite short in some cases, so that no operation for division of nerve fibres is likely to be successful. I have seen posterior root section fail on more than one occasion to relieve the pain in post-herpetic neuralgia of the trunk, and it has been suggested that in such cases the pain may be kept up by some morbid process connected with the herpes affecting the grey matter of the posterior horn, what may be called a posterior poliomyelitis. It is probable therefore that the operation of chordotomy offers a better chance of arresting post-herpetic pain than does posterior root section. Chordotomy is, however, a very delicate operation to perform, and by no means easy, and there is, moreover, an added risk to consider of partial paralysis of the legs being produced by the pyramidal tracts being wounded at the time of the operation.

For the surgical treatment of post-herpetic trigeminal neuralgia a Gasserian operation may be undertaken and the sensory root of the fifth nerve divided and avulsed behind the ganglion. Just as in the surgical treatment of paroxysmal trigeminal neuralgia, it is possible in the

majority of cases to reach the ganglion by a needle and inject it with alcohol, a procedure which it is difficult if not impossible to accomplish with the spinal root ganglia. I have thus treated several cases of persistent pain after trigeminal herpes, causing total fifth nerve anæsthesia and cure of the exhausting pain.

The method of procedure for injecting the Gasserian ganglion with alcohol in cases of post-herpetic trigeminal neuralgia is precisely the same as that described in the treatment of chronic paroxysmal trigeminal neuralgia (p. 205).

Bi-lateral Herpes.

Bi-lateral zoster is a great rarity, though occasionally cases have been described, but I am not aware of any published cases in which the same root areas on the two sides of the body have been attacked at the same time; indeed it is an old superstition with regard to shingles that if the rash were to spread right round the body the patient would die, a safe threat, because it is practically unknown to occur. The nearest I have seen to a complete girdle of zoster was in a woman suffering from splenic leukæmia who developed shingles as the result of prolonged arsenical treatment.

In her case two posterior root zones were involved, the seventh dorsal on the left side and the eighth dorsal on the right, so that there was a half girdle of vesicles on the left side coming round to the region of the xiphi-sternum, which just met the upper level of a similar band of herpetic vesicles on the right side of the trunk.

It will often be noticed that herpetic vesicles may cross the middle line for a distance of about a quarter of an inch, or even more in zoster affecting the trunk. Trigeminal zoster I have never known to be bi-lateral, and in the large majority of cases it affects the first or ophthalmic division only, so-called frontal herpes. I have never seen trigeminal zoster affect the tongue, though I have seen a profuse zoster rash on the side of the tongue in geniculate herpes. Zoster should not be confounded with the febrile

herpes in which the outbreak of vesicles is liable to occur round the lips and nose on both sides in cases of pyrexia, such as may occur with common colds, influenza, pneumonia and the like. This form of herpes is not painful at its onset, it does not leave scars, nor is it followed by subsequent neuralgia.

Complications and Sequelæ of Zoster.

When the actual attack of herpes zoster has been seen, or when the characteristic scarring along the area of distribution of a posterior spinal root is clearly visible, there is no difficulty in recognising the persistent after pain as being post-herpetic neuralgia. Yet it should be remembered that herpes zoster may result from irritation of a spinal root and the attack of zoster and the appearance of the rash may be evidence of a deeper lying progressive disease, such as spinal caries, secondary carcinoma of the vertebræ or osteitis deformans.

Slight herpes trigeminalis may also result from alcohol injection of the Gasserian ganglion in cases of ordinary paroxysmal trigeminal neuralgia.

The appearance of the rash on the anæsthetic area is precisely like that of an ordinary zoster, but is not accompanied by any degree of pain, though the lips and cheek will show some swelling. This form of zoster is doubtless due to the irritating effect of the alcohol on the ganglionic nervous tissue, and it may be safely ignored. When there is much scarring as the sequel of zoster it will be found that the scars are anæsthetic to pin prick, though usually the skin between the actual scars will have normal sensation. In some cases, however, of trigeminal herpes there will be dense anæsthesia of practically the whole distribution of either the first, second or third divisions. In old standing cases pigment will be present round and between the scars. In addition to persistence of the pain after herpes another chronic and irritating sequel may be *hyperidrosis.*

For example, a young clergyman, *æt.* 29, had an acute

attack of herpes zoster of the third division of the left trigeminal nerve; the pain was very severe for some weeks, but it passed off completely after four months, leaving typical scarring and anæsthesia on the lower lip, chin and temple. When I saw him four years later, in 1913, there was still considerable anæsthesia over the inferior dental and auriculo-temporal branches of the fifth nerve, and his complaint was of excessive sweating over this area on the left side, especially after eating. There was also some wasting of the masseter and temporal muscles, though he said they were improving, but there was no loss of sensation on the tongue. He mentioned that sweat often poured down on to his clothes from the left side of his face, which was a great nuisance to him in his social duties.

On testing him with food, flushing of the anæsthetic area was seen within fifteen seconds, and after sixty seconds beads of sweat were visible. This was a much more rapid response than in a case of hyperidrosis of the face in syringobulbia which I tested in the same way. This man noticed that pungent food such as vinegar or cheese provoked excessive sweating from his face, especially on the right side, but on testing him by giving him cheese to eat a lapse of several minutes occurred before sweating was observed.

CHAPTER XIV

MIGRAINE AND ALLIED NEURALGIAS

Migraine

Classical migraine, or hemicrania, is a one-sided headache, commonly ushered in by various warnings, particularly visual spectra such as scintillating scotoma and fortification figures, showing all the colours of the spectrum, or simply a sudden onset of hemianopia may occur, lasting from twenty minutes to half an hour. In some persons the visual phenomena are less elaborate, slight blurring of the vision or cobweb figures, or moving points of light like fireflies are noticed. With some rare cases the visual warning may be psychical, some scene suddenly appearing before the patient, perhaps enveloped in coloured light, the same scene being constant and never varying; these psychical auras are usually accompanied by a state of reminiscence in which the patient's memory seems to throw back to some long-forgotten incident which can never be definitely remembered, but may appear to suggest some occurrence in a previous existence. This mental state of a warning before the attack accompanied by reminiscence is strikingly similar to some warnings in the petit mal variety of epilepsy, the chief point of difference between them being the infinitely shorter period of time occupied by the epileptic aura. Curious dreams occur at times in migrainous subjects, especially frightening dreams of fire, and hemianopic dreams may recur from time to time, like the psychical aura, in which a scene is presented, such as a house and garden, in which only half of the house and garden may be visible.

Other warnings in migraine are tinglings and pins-and-

needles sensations running along the lips and tongue on one side, sometimes spreading into the arm and hand on the same side, though rarely affecting the leg.

Temporary weakness and numbness of the hand and arm may accompany this state and persist for twenty to thirty minutes, while a dazed, muddled feeling obscures clear thinking, and when the symptoms are right-sided actual aphasia may be present in varying degrees, wrong words being used, with similar inability to write and to read with understanding. Some sufferers from migraine frequently notice what may be called anticipatory warning, in a sense of physical fitness or well-being the day before the attack, while others may notice the appetite unusually keen. Indeed during some headaches of migrainous type I have found the appetite to be described as voracious, even in spite of recurrent vomiting.

From twenty minutes to half an hour or so after these warning phenomena the headache commences as a gradually increasing pain on one side of the head, often beginning as a pain over the eyebrow or behind the eyeball, through the temple and spreading over the back of the head and down the neck.

The scalp may become intensely sensitive and intolerant of hat pins or brush, and the pain may reach an agonising intensity lasting for hours until sleep, perchance, relieves or vomiting occurs. Sometimes the headache is continued into the second or even the third day. The frequency of recurrence of these migraine attacks varies enormously. They may recur about once a month or even much oftener, even two or three in a week, while other sufferers from this complaint may have them at very much longer intervals, a year or more separating the attacks.

The reason for their appearance is by no means always obvious, though much worry and mental strain are often noticed to aggravate the frequency of the attacks. Their origin can often be traced into earliest infancy, when they may be called bilious attacks, the severity of the vomiting causing regurgitation of the bile from the duodenum into

the acid-bearing gastric cavity, so that the yellow bile is oxidised to a green colour.

A family history of sick headaches or bilious attacks is frequently met with, and other varieties of family neuropathic tendency may also become evident.

This is not the place to write a treatise on the classical form of migraine, a subject which is worthy of separate treatment, but a short description has been given as an introduction to other forms of neuralgia which are really migrainous in type. These neuralgias I am accustomed to speak of as migrainous neuralgia, and one of the most striking and deceptive forms it may assume is *abdominal migraine*, a form of gastric crisis with severe abdominal pain and recurrent vomiting which may last for a day or two. This is sometimes met with in childhood but may persist throughout life, and may alternate with facial neuralgia. An instance of the latter will be described under ciliary neuralgia (see p. 307).

Migrainous Neuralgias.

Although I include the previously described types of daily recurrent supra-orbital or temporal neuralgia as migrainous in origin, more characteristic examples of migrainous neuralgia are those in which the attacks occur at frequent intervals, such as once in two or three weeks or oftener.

Sometimes the attacks are as frequent as three or four in a week, the patient being hardly ever free from pain, and in a few cases that I have met with excruciating pain in the temple and side of the head or in the cheek might last for an hour or even less, but would be repeated three or four times a day, the daily paroxysms continuing for years. Cases such as these may easily be mistaken for paroxysmal trigeminal neuralgia, and I have known treatment by alcohol injection and even gasserectomy performed under this mistaken diagnosis. They may be distinguished by the absence of any sensitiveness of the skin of the face or nose to light touches, by the accom-

paniment of nausea in some cases, or even vomiting, and by the fact that eating, talking or handling the face does not bring on the paroxysms, which last from fifteen minutes to an hour or more, as compared with the short spasms of half a minute or so in trigeminal neuralgia. With migrainous neuralgia there may be a history of ordinary migrainous sick headaches in earlier life up to the time of the appearance of the substituted migrainous neuralgia, the change in type often occurring after some emotional or physical strain such as bad news or childbirth.

I distinguish migrainous neuralgia from migraine only for convenience of description, the pain being less of a dull or severe headache type, but more neuralgic and superficial, often spreading into the face. Sometimes the pain is supra-orbital in distribution, but perhaps more commonly it is located on the temple, three-quarters of an inch outside and above the outer canthus of the eye. Pressure on this point on the temple is intensely painful, the tenderness even persisting in some cases between the attacks. Migrainous neuralgia differs from migraine in never being preceded by the aura so familiar in true migraine, such as hemianopia, scintillating scotoma, psychical reminiscence, tinglings of mouth and lips or aphasia. Migrainous neuralgia too is only rarely succeeded by vomiting, though in some cases slight nausea is recognisable. Just as in true migraine the pain may alternate from one side to the other, yet more frequently than in true migraine the attacks are limited to one side or, if alternation is observed, the pain is always more severe on one side.

Alcohol injection locally into the tender spot at the temple rarely cures, nor indeed does destruction of the Gasserian ganglion always arrest the recurrence of the attacks. When the pain has started or is fully developed an injection of morphine is probably the only remedy which can arrest the pain. Analgesic powders containing pyramidon, aspirin, phenacetin and numerous other varieties are not of much service here. Treatment, however,

between the attacks, on the same lines as are sometimes serviceable in true migraine, may notably diminish the frequency of the paroxysms, or may even cause their total disappearance. The one drug that I have found of real value in the treatment of migraine and migrainous neuralgia is luminal, a phenyl derivative of veronal. This drug, like veronal, may be combined with sodium, forming luminal-sodium, containing ninety per cent. luminal, a soluble salt comparable to medinal, the sodium salt of veronal.

It is, however, a more powerful drug and must be given in small doses, three-quarters of a grain given every night often being sufficient to check the onset of the attacks, and later the dose may be reduced to half a grain twice a week. The value of luminal in migraine and migrainous neuralgia is considerably enhanced in those cases in which sleep is difficult, the hypnotic action of the drug being of special benefit here, and as much as one grain or one and a half grains twice daily may be necessary at first to produce the desired effect. Migraine resembles epilepsy in being a paroxysmal neurosis, and there are many points of similarity between the two diseases which may indeed alternate in the same family and even in the same individual. The value of luminal in the treatment of many cases of epilepsy which have failed under bromide treatment is gradually becoming well known, and it is because of the analogies between epilepsy and migraine that I have employed luminal in the treatment of the latter disease.

A lady of 44 I saw in 1923 had suffered since she was a schoolgirl with neuralgia affecting one or the other side of the face and eye; the pain would begin in the eye and under the eyebrow and would spread to the temple. This pain would continue for five hours and then disappear for half an hour, returning suddenly "like a blow" on the other side. In other attacks the pain might last a whole day on one side and then attack the other. Of late years the pain has begun in the

morning and lasts until the early evening. Only rarely does nausea accompany these attacks. With a bad attack she has to lie in a darkened room, but the pain is not aggravated by movements of the face, though it is apt to be brought on by any extra worry and exhaustion. She has never found drugs of much use, but has noticed that she is always free of the neuralgia during the months of gestation. This immunity from migraine I have frequently observed in pregnant women.

Supra-orbital neuralgia is frequently a substituted migrainous pain.

Nine years ago I saw a doctor, *æt.* 67, who had suffered from typical migraine all his life, and even then still had frequent attacks of scintillating scotoma, often two or three times in a day. For two years he had been suffering every morning from violent left supra-orbital neuralgia, "awful pain" commencing almost suddenly within two or three minutes of waking, and lasting for an hour, though the attack might recur again in the afternoon. Alcohol injection of the supra-orbital nerve gave considerable relief, though it did not cure the attacks.

The pain in migrainous neuralgia frequently spreads into the face and jaws, so that it may be mistaken for paroxysmal trigeminal neuralgia, and many cases have been injected with alcohol under the wrong diagnosis of chronic paroxysmal trigeminal neuralgia. Thus, a lady, *æt.* 36, had suffered from severe attacks of hemianopia for many years, and for the last ten years the attacks of pain ran round the cheek and lower jaw to the back of the head, followed by vomiting. The neuralgia began in the cheek near the ala nasi, and spread round the eye, with a bumping pain on the top and back of the head where it would be most severe. No movements of the face such as eating or talking would start the pain, but fatigue, worry, or a cold draught would induce the attack. As her pain was always on the right side, it had been mistaken elsewhere for neuralgia of the fifth nerve, and an unerupted right lower wisdom tooth had been removed

without benefit. Injection of the Gasserian ganglion also had been performed but the migraine persisted.

Migrainous supra-orbital neuralgia may be bi-lateral and alternate from one side to the other in the same day or in different attacks. A common form of this neuralgia is its onset in the early morning sometimes within a few minutes of waking or soon after rising, lasting all day until the early evening. This pain may be accompanied by nausea or even vomiting, and is to be distinguished from the supra-orbital neuralgia associated with sinus infections. The long history of recurrent attacks, especially if associated with nausea, and the absence of evidence of sinus infection, will usually make the diagnosis clear. Although these attacks are truly migrainous in type, it is an undoubted fact that alcohol injection sometimes brings considerable relief for a time, for as long as two or three years or more. This result of injection of the supra-orbital notch may be compared with the similar relief produced by the injection of the infra-orbital nerve in cases described as ciliary neuralgia (see p. 307). Regular periodic migrainous supra-orbital neuralgia may commence at the menopause in women who have never previously suffered from headaches, the pain over eyebrow and temple being agonisingly severe, and recurring regularly every three or four weeks, apparently replacing the lost menstrual periods. The pain may last from four or five hours to twenty-four hours or more, and no nausea or sickness may occur. The neuralgia is likely always to commence on the same side, though occasionally it spreads across the forehead to the other side. Slight tenderness may be noticeable over the supra-orbital nerve at the notch, and a successful alcohol injection at the notch, producing supra-orbital anæsthesia, is likely to arrest the attacks. Drugs are mostly useless for this pain, but a mustard leaf to the neck, Frozoclone to the forehead and the inhalation of a few drops of Chlorylen may occasionally give relief.

In many cases of migrainous neuralgia the pain affects

especially the temple on one side, and there may be an intensely tender spot on pressure over the point about three-quarters of an inch slightly behind and slightly above the outer canthus of the eye.

A New Zealand sheep farmer, *æt.* 26, when staying in this country, noticed one night when getting into bed a sharp, heavy pain in his right temple as if he had been struck, the pain keeping him awake for hours.

Next morning the pain had gone, but every day of his life since the same pain returned between 4.30 and 5 in the afternoon. There was occasional nausea, but never vomiting. No treatment had ever done him any good, though exposure to cold always made it worse. This he noticed especially during the winter months when in the trenches during the War. His father had for years suffered from some form of pain in his head, which caused him to commit suicide at the age of 60.

The influence of cold upon migrainous neuralgia is very striking, and I have often thought that there is some association between some forms of chronic rheumatism and migraine. In 1921 I saw a woman, *æt.* 47, who for four years had suffered almost continuously from pain in the left supra-orbital region. At the commencement, four years previously, she had the pain daily for eight hours from January until April, being free for the remainder of the year, with a similar recurrence during the same months the following year. In 1919 the attack was more severe, and ever since then until I saw her she had daily pain from 12 noon until 5 in the afternoon, with never a day free. The neuralgia affected the left side of her forehead and the side of the head as far back as the level of the ear, and extended along the left side of the ridge of the nose, thus corresponding fairly closely with the ophthalmic division of the fifth. Alcohol injection of the supra-orbital nerve, though it produced typical anæsthesia and numbness of the forehead, did not relieve the pain. I then injected the Gasserian ganglion through the foramen ovale, producing total trigeminal anæsthesia,

which was still complete half an hour later, though when examined three days afterwards a good deal of sensation had returned over the first and second divisions of the nerve. The neuralgic attacks ceased from the time of the injection for some weeks, though they later recurred in a very much slighter form, being described then as a crawling and pulling sensation for some hours in the afternoon instead of the hammering pain which she formerly had.

Although in characteristic migraine visual spectra and fortification figures so commonly precede the headache, yet in migrainous substituted neuralgia it is very rare for visual phenomena to occur. The following case, however, is exceptional. A Catholic Sister, *æt.* 53, had suffered from bilious headaches all her life, associated with visual spectra of zigzags, with vomiting. For the past twelve months she had developed periodic attacks of numbness in the face as though the right eye was not there. This was followed by gradual loss of vision of the right eye, which became complete, even to loss of perception of light, the sight gradually returning after about eight minutes, the whole warning lasting about twenty minutes, and then succeeded by headache in the right temple and around the eye, with nausea, but no vomiting. During the intervals between the headaches pressure on the temple with the blunt end of a pencil would elicit tenderness and pain resembling the commencement of a neuralgic attack. Alcohol injection of the tissues at the tender spot down to the bone gave an excellent result. Ten months later no further attack had occurred.

Periodic Migrainous Neuralgia.

Migrainous neuralgia, like migraine, varies widely in the frequency of its attacks. These may be quite regular, occurring roughly every few weeks, or they may occur daily for weeks on end and then disappear for many months. In some cases the attacks are invariably on one side of the head only, though in the majority the

pain may attack either side, but even then severe neuralgia is usually limited to one side, the patient always knowing the attack will be comparatively slight when the pain is felt upon the other side. In many cases the pain is located principally in the temple, being likened to a knife being driven in through a point between the outer canthus of the eye and the hair line; this spot is often intensely tender to pressure, while the scalp may be exceedingly sensitive to a hairbrush, hairpins and other manipulations. Pain may spread over the forehead and one side of the head and down the neck, and not infrequently it radiates into the face, affecting both upper and lower jaws and the side of the neck. It is this latter type of case which is so often mistaken for paroxysmal trigeminal neuralgia. There should not really be difficulty in distinguishing the two conditions, for the pain never runs along the lips and tongue as in tic douloureux, nor is the pain started by movements of eating or talking, or handling the face. Moreover, trigger zones are never present in migrainous neuralgia, that is to say, light touches on the lip or side of the nose or cheek will not start paroxysms of pain as in tic douloureux. The painful attacks in the migrainous form are never momentary, or lasting only a minute or two, as is characteristic of trigeminal neuralgia, while, lastly, many cases of migrainous neuralgia are accompanied by nausea and may even end in vomiting, which is never seen in paroxysmal tic.

Although the pain in the large majority of cases of migrainous neuralgia continues for several hours, yet occasionally the attacks are much shorter, lasting only from ten minutes to an hour, and may recur several times within the twenty-four hours. It is these more frequently recurring short attacks which closely resemble the chronic paroxysmal tic, but even then there should be little difficulty in distinguishing them. For example a woman I saw in 1923, *æt.* 39, had for fourteen years suffered from recurrent attacks of right facial neuralgia, the paroxysms of pain affecting the right cheek and upper

and lower teeth, beginning suddenly, though not started by eating or other movements of the face. She described the pain as being like a "gathering," and it would spread over the right side of the back of the head and half the neck, lasting altogether about ten minutes, several attacks sometimes occuring during the night. The pain was so severe that at one time the question of tumour in the jaw or pharynx had been considered at one dental hospital that she attended.

Another somewhat similar example I saw in a lady of 72, who had suffered since childhood from intermittent neuralgic headaches, and whose mother had suffered from severe sick headaches. Since her change of life at the age of 45–50 she had suffered much more severely from neuralgia, the pain starting on the left side of the top of the head and spreading into the face and both upper and lower jaws on the left side. The pain also spread down the arm from the left shoulder to the hand; it worked up gradually and disappeared slowly, and was not started by any movements of handling the face or eating. The attacks of pain would last for half an hour, or even less, and she often had as many as eight or ten attacks in a day. No vomiting or even nausea occurred, though she complained much of flatulence, both gastric and of the bowel during an attack, the relief from flatus appearing to assist the disappearance of the pain. There was no arteriosclerosis nor any evidence of sinus infection, and being edentulous dental causes could be excluded. This lady was sent to me as a case of trigeminal neuralgia for alcohol injection, and the resemblance of this case of migrainous neuralgia, with numerous short daily attacks, to paroxysmal trigeminal neuralgia is superficially close.

In other cases the migrainous neuralgia may occur daily at about the same time and last for several hours for a period of a few weeks only in the year, and may recur annually at about the same time.

In 1907 I saw a man, *æt.* 44, who every year since he was 17 years old had suffered for some weeks in the autumn

from neuralgia affecting the right temple. Usually he was awakened about 3 a.m., the pain appearing to start quite suddenly as a sharp stabbing in the temple behind the outer canthus of the eye. The pain appeared to throb slowly, though not with the pulse, and became so severe that he said he was almost crazy with it and could not stand without staggering. The attacks lasted two or three hours, and often the neuralgia disappeared quite suddenly, though sometimes a very severe attack would make him vomit, after which the pain ceased. On some occasions he had two or three attacks in one day, and he was also subject to occasional left-sided sick headache, though he said the pain in that form was quite different and not so intense.

Migrainous neuralgia, like migraine, may occasionally be accompanied by excessive hunger, though visual spectra are never met with in this substituted form of migraine, but, as is well known, hemianopia, scintillating scotoma and other forms of visual spectra are not uncommon in ordinary migraine. In only one case have I known temporary amblyopia of one eye accompany attacks of migrainous neuralgia around the eyeball (see p. 301).

A lady, *æt.* 36, whom I saw in August 1922, had been subject since early childhood to left-sided headaches, often preceded by a tired feeling.

Of late years the pain had been located over the left supra-orbital notch, spreading over the forehead to the crown of the head and downwards into the left cheek and temple. She used to get these attacks about once a week, as a rule waking with the pain, which would last from one to two days. She always noticed excessive hunger with the headaches, and at the same time nausea but not vomiting. This combination of voracious appetite together with nausea and sickness in migraine I have seen on several occasions, though the majority of sufferers from migraine prefer to take no food during an attack. This patient had no visual spectra with the headache, but her youngest brother was subject to typical migraine.

The distribution of her pain being largely over the territory of the supra-orbital nerve decided me to try the effect of injecting that nerve at the notch with alcohol, as luminal, pyramidon, belladonna and other drugs had failed to relieve the recurrent attacks. In November 1922 I injected her left supra-orbital notch, getting typical supra-orbital nerve anæsthesia over the forehead to the top of the head; this arrested the recurrent attacks of pain completely, no further attack of neuralgia having occurred fourteen months later.

Another instance of uni-lateral migrainous neuralgia involving the eyeball, and accompanied by voracious appetite was sent to me in 1922, as a case of trigeminal neuralgia. A young man of 25, when aged 19 began to suffer from daily attacks, for three or four weeks every year, of severe pain in the back of the right eyeball and forehead, with lachrymation and reddening of the eye; this would last for an hour or two, no nausea, but voracious appetite accompanying the neuralgia. During the last two years he had had similar attacks of pain behind the left eyeball, and they would then transfer and become severe on the right side. He had never been subject to sick headaches, but his mother suffered from periodic headaches and was neurotic, and his eldest brother was neurasthenic. This was, no doubt, a case of migrainous neuralgia, and he received much benefit from taking luminal, having complete relief for thirteen months, when the attacks started again after riding. This recurrence of daily attacks was arrested by anticipatory injections of morphine and hyoscine given in the early morning.

Examination of the accessory sinuses in these cases is always negative, and though some might claim this and other cases to be instances of Sluder's or spheno-palatine neuralgia, the alternation of the attacks on the two sides, the family history and the accompanying voracious appetite definitely fix the attacks of pain as migrainous in nature.

The *pathology* of migrainous neuralgia is necessarily

mainly speculative. Its association with migraine is suggestive that the cause in each case is similar in type. In migraine many points add weight to the view that a fundamental part of the process is a vasomotor spasm of cortical and possibly meningeal vessels. When this affects the territory of the posterior cerebral artery, visual spectra such as hemianopia, fortification spectra, etc., may be looked for, while more anteriorly vasomotor spasm may affect the motor centres, when tingling around the lips and tongue, aphasia and even weakness of the arm and rarely also of the leg may occur as a temporary phenomenon. In these stages which may last for twenty minutes to half an hour there is no headache, but as the prodromal symptoms pass away severe headache quickly develops, perhaps due to vaso-dilator action and cerebral congestion.

The vasomotor spasm may rarely proceed to obliteration of the vessel by thrombosis, and I have known permanent hemianopia to result on four occasions, while, in another, thrombosis of one posterior inferior cerebellar artery happened during an attack, causing intense vertigo which kept the patient in bed for many weeks. The pain of migrainous neuralgia being referred to the eyeball, supra-orbital nerve and face, may perhaps be explained by the vasomotor spasm in these cases affecting the more anterior regions of the meninges, and thus causing referred pain over areas of the trigeminal nerve through recurrent meningeal branches of the three divisions of this nerve. Possibly on account of this anterior area of the meninges being involved in migrainous neuralgias, we have an explanation of the practically invariable absence in them of visual spectra, which are associated with the posterior occipital vascular supply.

Sluder's Neuralgia.

Sluder [1] has drawn attention to chronic neuralgia around the upper jaw at the root of the nose, in and about the eye and sometimes spreading to the region of the zygoma

[1] "Headaches and Eye Disorders of Nasal Origin," St. Louis, 1918.

and mastoid and the back of the head. This pain may be chronic, with acute exacerbations, or it may be intermittent like migraine, or the neuralgic spasms may be sudden and tic-like.

Sluder ascribes the pain to the spread of nasal sinus infection to the spheno-palatine, or Meckel's ganglion. Quite possibly he might include as examples of spheno-palatine neuralgia those cases of chronic neuralgia of the upper jaw that I have described (p. 226) and other cases which I describe under the headings of ciliary neuralgia, supra-orbital and migrainous neuralgia, and certain cases which may be classed as psychalgia. The pathology of all these cases is very speculative and perhaps impossible to ascertain with absolute certainty, though clinical examination of Sluder's cases lends little support to his thesis of infection of the spheno-palatine ganglion. Undoubtedly the treatment he advocates of cocainisation of the ganglion through the upper nasal passages has at times arrested the pain, but extirpation of Meckel's ganglion completely failed in other cases to relieve the neuralgia.

Ciliary Neuralgia.—Under this heading I include those cases of migrainous neuralgia in which the pain is referred locally to the eyeball alone or to the back of the eyeball, and I label them as ciliary more for descriptive purposes than with any certainty of their pathology.

Indeed I am convinced that these cases are a form of substituted migrainous neuralgia, and are therefore to be looked on as a spasmodic vasomotor neurosis rather than due to local disease of any ganglion or nervous structure. Illustrative cases with the effects of treatment that was tried will be the best means of gaining a comprehensive survey of this difficult type of neuralgia.

A man, *æt.* 47, had his first attack of neuralgia in January 1917, when in the trenches in front of Beaumont Hamel. The pain struck him suddenly across the left temple and forehead, and lasted for three-quarters of an hour, like an "electric battery," while his face became

flushed and he felt faint. The neuralgia recurred thrice daily, at about eight hour intervals for six weeks, and then disappeared entirely for two years. Ever since his first attack he had left cervical sympathetic paralysis, there being slight ptosis, with a small pupil. In subsequent attacks the neuralgia began on the left side of the top of the head, the eyeball being very painful and red, and the pain spreading down the nose into the left side of the cheek. The second attack lasted for six weeks, while his third attack did not occur until three years later, the pain awaking him shortly before 5 a.m. His face became sore during the attack, but there were no physical signs except the sympathetic palsy referred to.

The only positive clue to the pathology of this case is the sympathetic paralysis, which, he said, came on with the first attack. This could not have been an inflammatory lesion of the ciliary ganglion as the pupil would then have been dilated from paralysis of the constrictor pupillæ, and diplopia from involvement of the branch to the inferior oblique would probably occur. I have seen this syndrome associated with intense pain at the back of the eyeball, which lasted for many days in a man, *æt.* 50, as a result probably of rheumatism, the only relief obtained being after the administration of salicylates. The neuralgia never recurred in this case nor would one perhaps expect it to do so as it was due to a definite neuritis of a nerve centre.

A characteristic example of migrainous ciliary neuralgia in which the pain was limited to the eyeball alone I saw in a girl, *æt.* 12, in March 1914. Ever since infancy she had been subject to bilious attacks, one or more every week, and liable to be brought on by any excitement. On waking in the morning she would feel unable to eat any breakfast, then pain would commence in the left eyeball, which became intensely severe and might last from ten to thirty hours, keeping her awake at night. The pain was limited to the eyeball only, and was not accompanied by any visual spectrum or blindness.

She got some mitigation by keeping her hand pressed on the eye, but no real relief until vomiting occurred. These attacks had recurred continually for many years without any spread of the pain beyond the eyeball. As I had had some success by injecting the infra-orbital nerve in similar cases, I did this in April 1914. A slight attack occurred the same day and a very severe one the following week, but for the next two years she was quite free from attacks. In reply to a letter of inquiry in March 1925 she wrote that between the ages of 14 and 21 any undue worry or excitement caused a return of the malady, but for the last two years she has been scarcely troubled at all. The beneficial effect of the alcohol injection of the infra-orbital nerve in causing disappearance of the attacks for two years appears indisputable, and the case is a very suggestive link between migraine and what I have called ciliary neuralgia, and so-called Sluder's neuralgia.

In 1911 I saw a man, *æt.* 40, who had suffered for three weeks almost constant pain in the left eyeball, having had only eight hours sleep during that period. His first attack had been three years previously, the longest interval between the attacks was two months, and they lasted from one to two weeks, the pain being continuous and limited strictly to the eyeball. For about two weeks before the neuralgia began, he used to get an aura of a bad smell like rotten straw in his nose on the left side. He could arrest the pain for half a minute by pressure over the infra-orbital and supra-orbital foramina. Examination showed nothing wrong with the eye or accessory sinuses, and I then gave him an alcohol injection into the infra-orbital nerve, causing anæsthesia of the usual distribution. The pain instantly left him, and nineteen months later he wrote in reply to a letter of inquiry that he had never had the slightest trouble since the injection was done, and that he had not had even a headache or any pain in his head.

A similar case was that of a man, *æt.* 60, a horse-keeper, whose first attack of right facial neuralgia occurred in

1914. Another occurred in January 1916, of short duration, and then a third in March 1919, the pain continuing almost without intermission until I saw him in August 1919, when butyl chloral gave him some relief. The neuralgia recurred violently in November, with acute throbbing in his right eyeball, the pain being extremely severe, and I then injected his infra-orbital nerve with alcohol. His relief from pain was complete and immediate, but since then I have not heard from him.

The following case clearly shows the close association between the previous case of eyeball neuralgia and migraine. A man, *æt.* 33, of high education, who had suffered from recurrent periodic headaches as a boy, one day in November 1912 when out shooting had sudden blinding pain in his right eyeball and at the back of the eye, which lasted for half an hour only, but returned twice daily for about the same period of time.

The neuralgia would start below the supra-orbital notch and thence pass into the eyeball and outwards to the temple and spread downwards into the upper jaw and gum. He said the pain in the eye "felt as though someone had pulled the eye out and was hammering it." After the intense crisis had passed away he felt as though oppressed with a "toothache pain" in the cheek.

These attacks continued for about a month twice daily and then irregularly for about another month, after which he was quite well until the following October, with the exception of slight tenderness outside the eye if over-fatigued. He then had a sudden return without any warning of similar pain, though even worse than before, recurring twice daily, waking him up after twelve at night, and again in the afternoon, being almost beside himself with agony. The pain was not influenced by jaw movements or by handling the face, and thorough examination by a dentist, by X-rays, a throat and nose specialist, an ophthalmic surgeon and a neurologist disclosed no cause for the neuralgia.

When I saw him in May 1914 the attacks had been

irregular for six months and were again becoming much more violent. I then injected his infra-orbital nerve with alcohol, and they ceased completely for fifteen months, when recurrence took place. With their onset he noticed definite nausea, and it seems to me clear that these attacks were really migrainous, being a substituted form of neuralgia in a migrainous subject. Probably the alcohol injection of the infra-orbital nerve caused relief and the cessation of the attacks through reflex shock, rather than as in true trigeminal neuralgia by causing anæsthesia through nerve-block.

Such a case as this of pain in the supra-orbital notch, eyeball, temple and upper jaw would surely be claimed by Sluder as a spheno-palatine neuralgia.

That a similar condition may be brought about by local injury is shown by the following case. A young man, *æt.* 24, on September 27, 1915, was hit by a rifle bullet through both cheeks, teeth being knocked out of upper and lower jaws on both sides and the tongue badly cut. A month later neuralgic attacks began in the eyeball of one side, and the pain afterwards alternated from one side to the other. During March and April 1916 he was having daily attacks, the neuralgia starting at the same time every morning about 7 o'clock and disappearing towards evening, the pain being limited to the eyeball. There was no nausea or vomiting. In August he had another bad bout of attacks, the pain in the eyeball being so severe that he could not control himself, it being unlike anything he had ever experienced. Each attack commenced with a warning of slight aching in the eyeball for about twenty-four hours.

The pain was independent of any surroundings or any mode of life, occurring both in France and England, and examination of the eyes and sinuses and of the nervous system disclosed no abnormalities. Injection of the infra-orbital nerve in December 1916 was not followed by immediate relief, though after four days the attacks diminished gradually and disappeared. With the exception of a bad attack of frontal neuralgia five years previously

he had not suffered from headaches, though he had been subject to occasional intense gastric crises, undoubtedly *abdominal migraine*. When a small boy he had these attacks every week with an intense agonising pain in the abdomen for seven or eight hours, followed by vomiting and tenderness of the abdomen, and he said the pain was as bad as the worst neuralgia. These gastric attacks have been less frequent since he became adult, occurring every two years or so. His last abdominal crisis was about ten days before I saw him, when he was quite free from the facial pain through taking pyramidon and heroin.

The lessons to be learned from this case are that a local injury to the face may precipitate attacks of a substituted form of migrainous neuralgia, and that these attacks of intense pain in the eyeball were truly migrainous is shown by their alternation from side to side. Undoubtedly also the abdominal crises of intense gastric pain lasting for hours and followed by vomiting which occurred weekly during boyhood, and at longer intervals when he became older, are to be looked upon as migrainous in nature. I have met three or four such cases of abdominal migraine, in another of which the attacks alternated with facial neuralgia, but I could imagine that it might be hard to persuade certain surgeons that a visceral neurosis and not organic abdominal disease was the cause.

Still another case of violent recurrent neuralgia in one eyeball occurred in a man of 51 with a neurotic family history, but no history of migraine. The first attack occurred quite suddenly in the left eyeball and around the eye, with lachrymation, and occurred daily in the evening for about three-quarters of an hour for four months. The pain then disappeared for nearly three years, when daily recurrences took place after considerable worries due to the War. Infra-orbital nerve injection broke the sequence of attacks, which soon disappeared and afterwards he was quite well for some years.

Treatment of Migrainous Neuralgia

The treatment of this form of neuralgia will vary somewhat with the distribution of the pain. Some cases will benefit notably from luminal in the same way as ordinary cases of migraine may do so; it is more especially the cases associated with sleeplessness and irritability which benefit from this drug. With the exception of luminal and aspirin, the best results are sometimes obtained in migraine with seven to ten grains each of phenazone and butyl chloral hydrate. In many sufferers all drugs are useless, though in them some relief may be obtained by a mustard leaf to the back of the neck, by painting Frozoclone on the forehead, or by inhaling the vapour of Chlorylen, twenty to thirty drops on a handkerchief.

Others in which the pain is located over the supra-orbital distribution, in the eyeball or in one fixed spot in the temple may occasionally show brilliant results with alcohol injection of the supra-orbital nerve, of the infra-orbital nerve or of the tender spot in the temple itself.

How such injection acts it is difficult to say; especially perhaps it is difficult to explain how severe pain in the eyeball, whose sensory supply is received from the ophthalmic or first division of the fifth nerve, can be instantly relieved by injection of the infra-orbital nerve, which is a branch of the second division of the fifth nerve.

Probably the alcohol injection acts in a reflex manner inhibiting the attacks of vasomotor spasm, and acting in this way it might not matter very much which branch of the fifth nerve is injected, though it is only natural to suppose that reflex inhibition of the vasomotor meningeal spasm causing the pain is most likely to be successful when that nerve is injected over whose territory the neuralgic pain is referred.

CHAPTER XV

GENICULATE NEURALGIA

Otalgia, or neuralgia referable to the geniculate ganglion and its sensory distribution, is a frequent accompaniment of that common form of facial paralysis, or Bell's Palsy, which may be the result of rheumatic neuritis, dental or antral sepsis, etc. The onset of such cases of facial palsy is commonly preceded by aching behind the ear for a day or two, the pain being sometimes severe and spreading forwards in front of the ear and into the upper part of the neck. This pain has nothing to do with catarrh or suppuration of the middle ear, and it is unaccompanied by any deafness.

The geniculate ganglion, so called from its being situated at a right-angle bend (*genu*, a knee) on the facial nerve within its bony canal on the inner wall of the middle ear, is not connected directly with the motor fibres of the seventh or facial nerve, but is comparable to a posterior root ganglion of the spinal nerves, or to the Gasserian ganglion of the fifth nerve, and the jugular and petrosal ganglia on the vagus and glosso-pharyngeal nerves. The ganglion is therefore connected with sensory fibres only, its main peripheral branches being the chorda tympani and the great superficial petrosal nerve. The central prolongation of the geniculate ganglion is known as the nerve of Wrisberg or pars intermedia, and lies between the portio dura or seventh nerve and the portio mollis or auditory nerve. This nerve of Wrisberg is comparable to the sensory root of the fifth nerve or to a posterior spinal root. The seventh nerve issuing from the stylo-mastoid foramen after winding round the ear hole is the nerve of the first gill slit or branchial cleft of fishes. While

its motor portion persists as the motor nerve of the facial muscles, its sensory portion has diminished in importance, its cutaneous distribution in man being merely vestigial, its sensory supply having been encroached upon by the trigeminal nerve in front and the cervical plexus behind. The chorda tympani carries the nerve fibres of taste to the anterior two-thirds of the tongue, and also salivary secretory fibres, while the great superficial petrosal, which becomes the Vidian nerve and joins Meckel's ganglion, probably conveys taste impressions from the posterior part of the palate. Ramsay Hunt has made a special study of the sensory supply of the seventh nerve, and by means of cases of herpes zoster and other clinical material has demonstrated that it supplies in part the tympanum and the postero-superior portion of the external auditory meatus, and the concha, or greater hollow of the external ear. Also by an anastomotic branch to the nerve of Jacobson of the glosso-pharyngeal, and Arnold's nerve a branch of the vagus, it aids in supplying the cleft between the auricle and the scalp at the back of the ear. The geniculate ganglion also sends nerve fibres to join the trigeminal system at the otic and Meckel's ganglia by means of the two superficial petrosal nerves. The lesser superficial petrosal is really a branch from the glosso-pharyngeal to the otic ganglion, and it receives a small branch from the geniculate ganglion. Both superficial petrosal nerves are joined by sympathetic fibres, the deep petrosal nerves to the tympanic plexus.

Geniculate neuralgia may appear spontaneously in rare cases as spasms of severe pain in and around the ear resembling tic douloureux, as in Clarke and Taylor's case (p. 251).

In other cases severe pain may last for weeks following herpes zoster within the ear and concha, or the pain may follow middle ear disease and a mastoid operation, or fractured base, or it may be accompanied by severe clonic spasm. In the case of a lady, *æt.* 58, who had suffered from clonic right facial spasm for five years, I injected

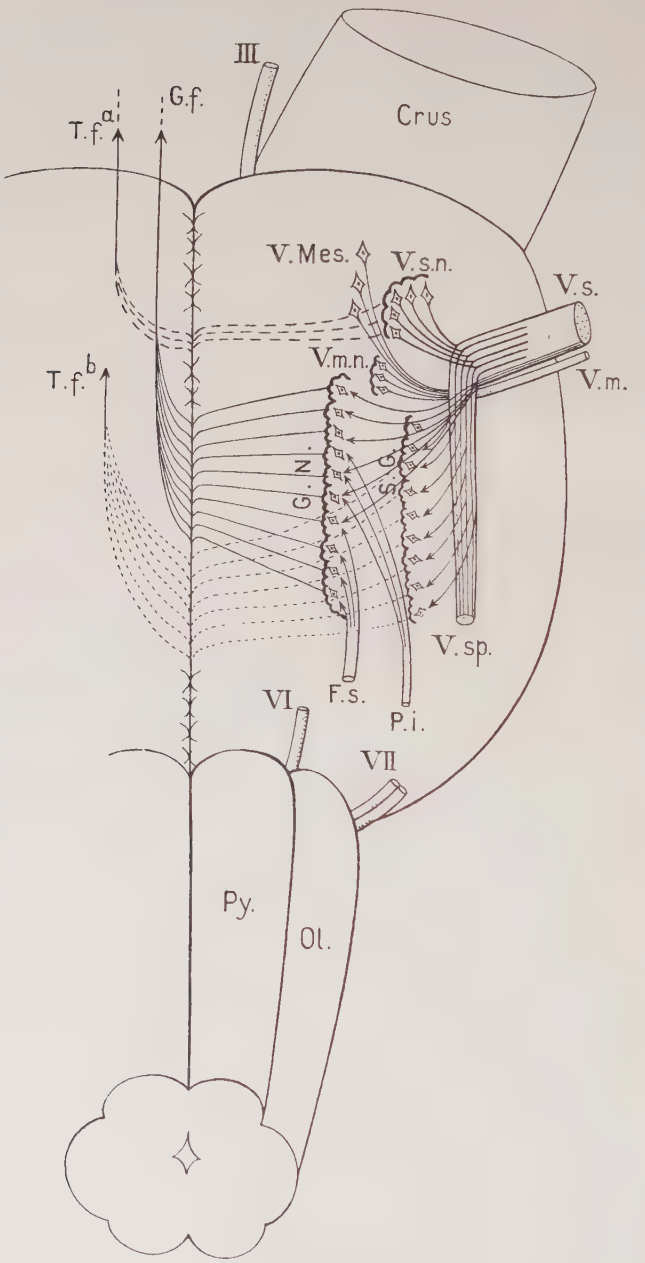

Fig. 40.

the stylo-mastoid foramen with five drops of strong alcohol, causing marked paresis of the facial nerve. Five days later she developed sharp neuralgic pain in front and behind the ear, also deep in the ear, and along the lower jaw and into the temple. This pain continued in paroxysms for a few days, preventing sleep. There was a severe freezing east wind blowing at the time, in January 1917, and possibly this was the cause of the geniculate neuralgia rather than the alcohol injection of the nerve; as in many other cases in which I have injected the facial nerve for clonic facial spasm I have never met with this complication of neuralgia.

In probably the majority of cases in geniculate herpes there is also facial paralysis, though, as Ramsay Hunt observed, this is by no means necessarily so. In Gasserian herpes and in herpes of spinal roots it is the exception to find motor paralysis and wasting associated with the herpes zoster and pain, though the anatomical relationship of fibres of the root ganglion is similar in each case. The following example of severe geniculate neuralgia as an accompaniment of herpes within the ear, without facial palsy, I saw in March 1911 in a medical man *æt.* 41. A week previously he had noticed a sudden sensation as if deaf in both ears for a time, leaving deafness and a sensation of fulness in the right ear, but no pain. He then

FIG. 40.—Diagram of ventral view of the intra-pontine connections of the left fifth nerve, to illustrate the path of the taste fibres.

V.s.	Sensory trigeminal root.
V.m.	Motor trigeminal root.
V.s.n.	Principal sensory nucleus of fifth nerve, or convolutio trigemini.
V.m.n.	Motor nucleus of fifth nerve.
V.Mes.	Mesencephalic nucleus of fifth nerve.
V.sp.	Spinal root of fifth nerve.
S.G.	Substantia gelatinosa or nucleus of spinal trigeminal root.
G.N.	Gustatory nucleus.
F.s.	Fasciculus solitarius, or ascending glossopharyngeal root.
P.i.	Pars intermedia Wrisbergi.
Py.	Pyramid.
Ol.	Olive.
T.f.a.	Trigeminal fillet (tactile and pressure).
T.f.b.	Trigeminal fillet (pain and temperature).
G.f.	Gustatory fillet.

had his right ear syringed, and immediately he was struck by intense pain in the right side of his head, over the whole distribution of the trigeminal nerve and over the back of the right side of his head and upper neck, the pain shooting and stabbing over the whole of this area, in addition to pain deep in the ear itself. The pain continued severely all night, so that he got no sleep and had to walk about. Next morning he noticed a sero-sanguineous discharge from his right ear, the pain being then easier. A doctor friend put some peroxide in the ear and again syringed it, which, as before, renewed the pain, which continued altogether for about three weeks. Deep herpetic vesicles were seen in the cleft at the back of the auricle at its junction with the scalp, and blebs were visible on the tympanum and also on the posterior upper wall of the meatus. There was marked loss of taste both to bitter and sweet on the right half of the tongue, and latterly some fibrillary tremor was visible in the right orbicularis palpebrarum. The paroxysms of pain continued daily for the next two weeks, getting worse at night time, the pain darting along the side of his head above the ear, with sensitiveness of the concha and tenderness of the scalp above the ear.

He noticed that his right pupil was dilated every morning, though it became equal to the other during the day. No anæsthesia was detected on testing, though he stated that the ear felt more dead at night, and a parchmenty feeling of numbness of the right auricle and of the skin in front, above and behind the ear, lasted for about a year. His taste was quite normal when I tested him two years after the illness.

This case illustrates the wide overflow of neuralgia over the trigeminal and cervical areas in an acute lesion of the geniculate ganglion. Noticeable also was the marked loss of taste, though there was no motor palsy, only fibrillary tremor around the eye indicating some irritation of the motor nerve.

In another case that I saw only a week previous to the

former, in a man of 42, right facial palsy developed without any pain, though two weeks later soreness within the ear was noticed, and severe pain began behind and in front of the lower half of the auricle, keeping him awake at night. Herpetic vesicles were visible on the membrane and the inner portion of the meatus, facial paralysis was complete and there was total loss of taste when tested with sweet and bitter on the right side of the tongue.

Again, in a man of 79, the ears were syringed for deafness six months previously; a fortnight later soreness developed within the right meatus, and ever since he complained of neuralgia within the right ear and of pain spreading over the auriculo-temporal area and affecting the right side of his throat. This was worse on swallowing, cold water being especially painful to drink. This patient was a well-marked example of oxycephaly.

A lady, *æt.* 31, as the result of a bicycle accident was unconscious for twenty minutes, bleeding from the left ear. She was completely deaf in that ear for a few days, with total loss of sense of smell and taste on that side. The loss of sense of smell was permanent, but the taste returned after several weeks. For the first two or three months she had pain in the ear, which then gradually left her for three months, when it returned severely, starting deep in the ear and spreading forwards on to the cheek and back of the jaw, and keeping her awake. The pain was not aggravated by movements but it never left her free for a whole day.

In this case there was probably a slight fracture of the base on the left side, and the initial loss of taste might be assumed to indicate a lesion of the pars intermedia Wrisbergi. There was, however, no facial paralysis, and the development of the neuralgia and the recovery of the deafness rather indicate a lesion in the neighbourhood of the geniculate ganglion.

Sometimes chill will set up an *acute geniculate neuritis* without producing herpes zoster, but followed by facial palsy.

In 1913 I saw a lady, *æt.* 48, who five years previously was exposed to a sharp chill, and the same night suffered with acute neuralgia in front of the right ear, spreading towards the temple and cheek, the pain being very severe and keeping her awake. Two days later complete right facial palsy appeared. For many weeks the pain never left her, but later disappeared.

Ever since she has been subject to recurrences of similar neuralgia in severe attacks lasting forty-eight hours, commencing with horrible screwing pain in front of the ear over the root of the zygoma, spreading above the eye and into the right cheek and gum, but never reaching the nose or lip, the lower jaw or forehead. Sometimes the pain spread into the back of the head, but never into the neck. A cold north-east wind would always start the neuralgia. Her dentist absolved the teeth, but she had extensive crown and bridge work, and her teeth have always been very sensitive, so that she was unable to wash them with cold water, and as a girl she suffered much from right-sided facial neuralgia.

This case resembles many cases of so-called rheumatic facial palsy, in that the pain preceded the facial paralysis for a couple of days, but it was much more severe and lasting, and the persistence of recurrent attacks of the neuralgia for several years is uncommon. Quite possibly dental infection may have played a part in this case, and I have for many years been convinced that some cases of facial palsy are due to dental sepsis infecting the geniculate ganglion or the facial nerve in its neighbourhood. The exciting cause of the chill, which was followed in this case immediately by the severe neuralgia, is absolutely comparable to the effect of chill in starting a dental neuralgia, where the nerve filaments are already affected by sepsis from caries, and the same sequence is also seen in many cases of typical paroxysmal trigeminal neuralgia.

In April 1914 I saw a young man, *æt.* 22, the son of a physician, suffering from acute geniculate neuritis, with facial palsy, and herpes zoster vesicles on the right border

of the tongue and on the soft palate. Two weeks before I saw him he complained of shooting pain deep in the right ear, keeping him awake for two nights. On the third day right facial paralysis appeared, preceded by a sensation of numbness on the right half of the tongue and also on the right lip. The neuralgia soon disappeared, except for a few sharp pains in the lobule of the right ear.

An outcrop of zoster vesicles next appeared on the right half of the tongue and on the soft palate. When I saw him, there was complete right motor facial palsy, with reaction of degeneration to electrical tests, and marked signs of herpetic vesicles along the right border of the tongue as far as the tip, and also on the right half of the soft palate, near its junction with the hard palate. No vesicles were seen in the meatus or on the membrane. Hearing was slightly diminished, both to aerial and bone conduction on the right side. No diminution of tactile sense could be detected on the tongue or palate, or on the lip or cheek. Taste sensation was completely lost on the right anterior half of the tongue, the recognition of saline and sweet being normal on the left side.

The crop of zoster on the half of the tongue and soft palate, associated with uni-lateral loss of taste and facial palsy, following pain in the ear, is a striking confirmation of Ramsay Hunt's thesis of the vestigial sensory supply of the seventh nerve, when taken in conjunction with other cases in which the zoster invades the tympanic membrane, the posterior upper wall of the meatus, the concha and the posterior portion of the auricle. The numbness complained of on the tongue and the crop of vesicles on the side of the tongue are suggestive of the presence of some tactile fibres in the chorda tympani, while the appearance of zoster on the palate at the same time is evidence of the nervous supply of the palate from the geniculate ganglion through the great superficial petrosal and the Vidian nerve, in exactly the same way as the chorda tympani supplies the tongue.

Care must be taken to distinguish zoster due to geni-

culate inflammation from trigeminal herpes which may accompany a geniculate neuritis with facial palsy. The two ganglia, Gasserian and geniculate, may be simultaneously attacked by the same inflammatory disease, probably infective, in which case there will be pain around and in front of the ear, associated with facial palsy. In addition to the geniculate herpes already described in the ear, and perhaps on the tongue and palate also, an outcrop of zoster vesicles over the area of one or more of the divisions of the fifth nerve will be accompanied by neuralgic pains in the trigeminal zone.

Thus a Catholic priest, *æt.* 41, in March 1924 developed some swelling in the right parotid region and neck, which was mistaken at first for mumps, and was accompanied by considerable pain in front of the ear. In a few days he developed complete right facial palsy, with an outcrop of profuse zoster on the right temple and side of the cheek and right side of the chin. No vesicles were seen in the ear, but a few occurred on the right side of the palate and also on the right side of the tongue in front of the anterior pillar of the fauces. When I saw him three and a half weeks after the first symptoms there was complete right facial palsy, with reaction of degeneration in the muscles and profuse herpetic scarring on the right side of the chin and face and temple.

A lady, *æt.* 59, had for thirty years suffered from pain in both ears since she was concussed by a fall from a carriage. For many years the pain affected only the right ear, spreading thence along the back of the lower jaw, and it was especially brought on by exposure to draught, so that she had to cover up her face carefully when driving in an open carriage. This may be looked on as a mild case of *bi-lateral geniculate neuralgia*, the result of a blow, and comparable to major trigeminal neuralgia, which may be similarly started by a blow upon the jaw.

The area of pain reference therefore in geniculate neuralgia is mainly in the ear, in front of and above the ear and along the back of the lower jaw, though when

very acute, especially at the onset of the disease, the neuralgia may overflow into the neighbouring trigeminal and cervical areas. There may be considerable hyperæsthesia of the scalp above the ear, and this area of severe pain and hyperæsthesia may be a valuable sign in the recognition of *acute mastoid infection*, when accompanied by intermittent pyrexia, even though there may be no otorrhœa, no œdema of the skin nor tenderness over the mastoid, nor any bulging of the posterior wall of the auditory meatus.

The pain is difficult to relieve, and may be so acute for some weeks that sleep is almost impossible. Syringing the ear is to be avoided, more especially with cold water. Hypodermics of morphine and hyoscine may be necessary, a quarter of a grain of morphine with $\frac{1}{200}$th of hyoscine, which may be repeated at intervals until the pain is relieved. In less severe cases combinations of pyramidon, aspirin and medinal will probably give relief. It is noticeable that the pain in geniculate neuralgia is often worse at night, the contrary being the usual rule in major trigeminal neuralgia. Chronic paroxysmal geniculate neuralgia, as in Clarke and Taylor's case, must be very rare. Two cases which I at one time thought to be of this character will be described as glosso-pharyngeal neuralgia.

I have seen severe paroxysmal facial neuralgia, probably of geniculate origin, follow immediately upon a mastoid operation, which produced facial paralysis on the same side.

A married woman, *æt.* 29, whose teeth were in perfect order, and who had never suffered from toothache in her life, developed attacks of paroxysmal pain in the left side of the face immediately after the onset of facial paralysis following a mastoid operation. There was always some pain behind the ear, and in the attacks, which occurred about once a fortnight, the whole side of the head and face became tender, so that she could not bear a hairbrush or hairpins, and there was considerable nausea. The pain was worse when she was worried, and in some points

the characteristics of the neuralgic attacks resembled migrainous neuralgia.

Cushing[1] described in 1920 a condition which he labels *Painful Tic Convulsif*, in which violent pain referred to the eye and face accompanied uni-lateral clonic facial spasm. In two of his own cases complete gasserectomy failed to relieve the pain, the facial spasms and neuralgia continuing unabated, and he also describes a third case in which Gasserian operation was done by another surgeon without relief. Of a total of five cases, two were relieved by a spino-facial anastomosis operation, one for six months and the other permanently. He infers that these cases may possibly be attributable to a lesion of the geniculate ganglion, as suggested by Ramsay Hunt. This certainly appears a probable solution, but there is a difficulty in diagnosing such cases correctly before trying alcohol injection or Gasserian operation. First, the pain began in the eyeball in two of his cases and spread into the face, the distribution of the neuralgia therefore not suggesting geniculate pain; secondly, convulsive spasms of the face to a considerable degree is a not uncommon accompaniment of many paroxysms of genuine trigeminal neuralgia, and careful observation will be required to distinguish a clonic spasm affecting all the muscles supplied by the facial nerve, and the reflex spasm of the face secondary to the flashing pain of neuralgia. Thirdly, clonic facial spasm may co-exist with and be apparently independent of trigeminal neuralgia; and, lastly, uni-lateral clonic facial spasm may be truly associated with paroxysmal trigeminal neuralgia of the same side of the face, as I have seen in quite a number of cases. The clonic spasms are usually not severe, but are quite definite and distinct from reflex facial contortion due to pain. That they are in some way dependent upon and associated with the trigeminal neuralgia is proved by the disappearance of the muscular spasms with the cure of the neuralgia.

In 1919 I saw a lady with trigeminal neuralgia of

[1] *Amer. Journ. of Med. Sciences*, 1920, Vol. 160, p. 157.

five years' duration which followed herpes zoster of the ophthalmic division. The pain, however, did not resemble post-herpetic neuralgia, but involved the second and third divisions of the nerve with attacks of sudden violent pain brought on by eating and other movements of the face, just as in ordinary neuralgia major of the fifth nerve. In addition she was the subject of clonic facial spasm of the same side which had existed for thirty years, affecting practically all the muscles of the face, but causing no pain, until the trigeminal neuralgia started five years before I saw her. Alcohol injection of the second and third divisions of the fifth nerve completely relieved her neuralgia, but the facial clonic spasms continued, until they were arrested a week later by an alcohol piqure of the facial nerve, at the stylo-mastoid foramen.

The following case rather more nearly resembles those described by Cushing as Painful Tic Convulsif.

A married woman, in 1918, when aged 46, noticed a throbbing, not painful, in the right side of her chin. Three years later neuralgic pain began at that spot, associated with spasms of the right facial muscles. When I saw her in 1921 I accepted the case as one of neuralgia major of the third division with reflex facial contortions, although a suspicious feature in the case was noted that the pain was not brought on by eating, talking or handling the face, but that if she put her finger into her right ear pain was at once referred to the right side of her chin. Injection of the third division at the foramen ovale was quite successful in producing dense anæsthesia of its distribution, but wholly failed to arrest the neuralgic spasms of facial tic. I saw her again three years afterwards, when the conditions remained unchanged, except for disappearance of most of the anæsthesia, the clonic right-sided facial spasms being intermittent and accompanied by moderately severe neuralgia. In May 1924 I injected the Gasserian ganglion through the foramen ovale, causing total ganglion anæsthesia, which was still complete six hours later, all neuralgia and facial spasm having ceased.

Fourteen months later, in July 1925, she was still completely free of the neuralgia, the ganglion anæsthesia remaining unaltered, and the clonic facial spasm had disappeared. This case, therefore, must be classed with my other cases of associated trigeminal neuralgia and clonic facial spasm, in which the two conditions appear to be dependent. The fact that the clonic facial spasm is always on the same side as the neuralgia is suggestive of some real association between the two conditions.

Although in such cases as Cushing has described it is probable that the irritant lesion is not on the peripheral side of the stylo-mastoid foramen, but perhaps at the geniculate ganglion or even central to this point, yet it is easy to understand that motor paralysis of the facial nerve produced at the stylo-mastoid foramen either by alcohol piqure or by section for the purpose of nerve anastomosis with the hypoglossal or spinal accessory, may reflexly arrest the neuralgic attacks which in painful tic convulsif always accompany the motor clonic spasms of the face.

Ramsay Hunt has suggested that clonic facial spasm is always the result, not of irritation of the motor fibres of the seventh nerve, but of sensory stimuli from a diseased geniculate ganglion affecting the facial motor nucleus centrally. This theory is attractive, but it scarcely explains the presence of definite facial weakness in cases of clonic facial spasm, which I have frequently observed, unless the geniculate disease involves the motor fibres also. Recurrent facial spasm I have also observed due to the downward pressure of a cerebellar tumour, which was cured by operation and removal of the growth.

Afferent stimuli from the geniculate ganglion may well be the cause of facial contracture and spasms which are such a common sequel to a partially recovered facial palsy. The fact that division of the facial nerve and its anastomosis with the spinal accessory nerve relieved the neuralgia, and of course the facial spasm, in two of the cases of painful tic convulsif referred to by Cushing is certainly strong evidence of the part played by the sensory fibres of the seventh nerve in these rare neuralgic facial spasms.

CHAPTER XVI

GLOSSO-PHARYNGEAL NEURALGIA

Glosso-pharyngeal Neuralgia.—In addition to chronic paroxysmal neuralgia affecting the trigeminal and geniculate systems there is a still rarer form of intense tic douloureux attributable to the glosso-pharyngeal nerve. I have met with three well-marked examples of this chronic tic, two lasting for many years, which in the severity and suddenness of onset of the paroxysms closely resemble trigeminal neuralgia. In one case in a lady *æt.* 87, the paroxysms had been continuing for over twelve years when I first saw her, and in another, a man of 50 had had neuralgia for five years, and in spite of treatment it continued to my knowledge for some years afterwards. In neither of these cases was there any sign of organic lesion in the throat or neck, and the long duration of the disease without the appearance of any sign of growth may be taken as sufficient evidence to exclude the possibility of malignant disease in the pharyngeal region.

In four other cases I have seen recurrent neuralgic spasms of glosso-pharyngeal distribution, in three of which malignant growth in the tonsillar region was present or had been previously operated upon, and in the fourth case lately under observation, a man of 54, I was suspicious of malignant disease. Thus in my experience there appears to be a form of glosso-pharyngeal tic precisely comparable to paroxysmal trigeminal neuralgia major, and also a reflex neuralgia of this nerve area secondary to malignant growth in the tonsillar or pharyngeal region.

Glosso-pharyngeal Tic.—A barrister when aged 45 was standing on Barnes railway bridge watching the University

Boat Race, and while eating a sandwich he quite suddenly felt an excruciating pain in the right side of the throat, lasting for a few seconds and reappearing in the throat on attempting to swallow. A few days later the pain spread into the right side of his face. Iodide of potassium was given and after six weeks the pain disappeared. A year later while playing golf the neuralgia reappeared, with severe pain on the top of his head, and he had recurrent epileptic fits every few minutes, each fit preceded by severe spasms of pain in the face. After a fortnight the fits disappeared, but returned eight months later for two months; he was then free again for two years when the fits again reappeared, each preceded by similar spasms of pain, and during the five years he had altogether about eight weeks of fits. When I saw him in 1910, five years after the first onset of neuralgia, he was having numerous spasms of pain starting in the throat on the right side, spreading down the side of the neck and in front of the right ear and back of the lower jaw. The pain never ran into the ear or into the gums or lips, though touching the auricle or concha or external auditory meatus would almost invariably start the pain. Four months before I saw him he had had three months of severe spasms of pain, day and night, and although he was having no fits at the time, he was trephined by a surgeon over the left motor cortex, the membranes and brain appearing normal. In September 1910 I injected his right third division of the fifth nerve at the foramen ovale, producing total anæsthesia and motor palsy of this division, with complete loss of taste on the right side of the tongue. The anæsthesia of the tongue, indeed, was so dense that he bit it badly on the right side without knowing it.

He continued to have scores of neuralgic spasms every day and they could generally be provoked by opening the mouth wide, by protruding the tongue towards the opposite side, by pressure in front of the ear, by rubbing the auricle, or by pricking the posterior wall of the meatus. Immediately after the attack of pain these areas were

unresponsive to stimuli for a few minutes, as in cases of trigeminal neuralgia major. He had a way of arresting the painful spasms by clearing his throat with slight coughs, and he said the pain caught him at the upper part of the back of his throat on the right side, behind the palate. The neuralgic spasms continued for four months, when he again had an intermission. He had had syphilis fifteen years before the neuralgia started, and before I saw him he had been extensively treated with mercury and iodide without any relief of the pain. Indeed the only drug which relieved the paroxysms in any way appeared to be bromide. He died in 1925.

Another case I have seen of this extraordinary neuralgic tic was an old lady *æt.* 87, living in very comfortable circumstances, whom I saw in July 1914 on account of neuralgic spasms which had begun over twelve years previously. She said she had been subject to sore throats all her life, and before the neuralgia began she had some form of emotional shock. Gradually from that date she began to have increasingly severe paroxysms of neuralgia beginning in the left side of her throat, the pain being very sharp, like stabs, affecting the left side of the throat and the left ear. She had no pain along the gum or left side of her head, and formerly she used to have agonies of pain on swallowing, but that became less severe.

Eating would not start the pain, but swallowing did. The ear was intensely sensitive to touch on the meatus and concha and in front of the ear, though the back of the pinna was not sensitive, and the whole ear was extremely dirty from her inability to bear it being touched. She slept fairly well, but was unable to lie on the left side. Injection of the third division of the fifth nerve with alcohol at the foramen ovale produced absolute anæsthesia of its whole distribution, with loss of taste on the left side of the tongue, but the neuralgic spasms and the tenderness of the ear to touch or pressure continued unabated.

These two cases resemble one another closely in the site of the pain, starting in the throat and being provoked

by the act of swallowing, and also in the zones of extreme sensitiveness of the front part of the auricle, of the concha and external auditory meatus, and to pressure in front of the ear.

A third case of glosso-pharyngeal tic I saw in August 1924. A ship's captain, *æt.* 53, six months previously had noticed a sudden jerk of pain from the root of his tongue into the drum of his left ear on swallowing. For ten days this recurred with the act of swallowing and then disappeared until the end of June, when similar pain started during the evening meal. Next morning when yawning similar pain attacked him in the left side of his throat and ear for three or four seconds, nearly causing him to faint with its severity. After this he suffered frequent spasms of most violent pain, started by eating and talking, but not by washing or handling the face. The pain seemed to start as a burning at the tip of the tongue, spreading to the root as a shooting pain and thence to the back of the jaw and ear, running into the concha. Though very suspicious that it was a glosso-pharyngeal tic and not trigeminal in origin, I injected the third division of the fifth nerve, causing total anæsthesia of its distribution, but the violence of the neuralgic spasms continued unabated, and they would start if the side of the neck below the angle of the jaw were rubbed, the pain referring to the back of the palate and ear. I then attempted to inject the glosso-pharyngeal nerve with alcohol near the jugular foramen, with the intention, in case of failure to arrest the pain, to have the nerve excised. The needle was inserted in front of the tip of the mastoid, and passed inwards and slightly upwards to a depth of one and a half inches. No anæsthesia of the pharynx resulted, but the attacks subsided after a fortnight, and no sign of the glosso-pharyngeal tic had returned when I saw him six months later.

Injection in such a region as the jugular foramen is not to be attempted by anyone without considerable experience of such injection work.

Somewhat similar neuralgia to the last case occurred in a clergyman *æt.* 65, who had a malignant growth removed from the upper part of the larynx in 1912. Three years later he began to have neuralgic pain in the region of the left tonsil, but on examination the throat surgeon found nothing. The neuralgic spasms continued, spreading deeply in the left ear, the paroxysms lasting about twenty seconds, the pain being very sharp and occurring about every quarter of an hour, or he might get some hours interval. He noticed that yawning started the pain, but swallowing did not affect it. When I saw him in 1918 the pain had continued for three years. He had had all his upper teeth removed, though X-rays showed nothing of importance. I found tenderness of the upper palatal fold on pressure, and twisting his neck to the left hurt him. Feeling suspicious of recurrence of growth in the tonsillar region I sent him to the throat surgeon for report. He found a small malignant adenoma on the left ary-epiglottic fold and another in the tonsil, both of which were then removed, and the neuralgia disappeared for a time. A year later he began to get stinging pains in his neck and lower jaw, also through the ear and on swallowing, and intense sensitiveness to pressure at the angle of the jaw. Drinking was excessively painful, and sneezing or blowing his nose caused stinging pain in the left lower jaw. Recurrences of growth appeared in the neck and became disseminated, and he died without the neuralgia having been relieved.

This case resembled the three cases of glosso-pharyngeal tic already described in the neuralgic spasm starting from the tonsillar region and spreading into the ear and neck, and in being started by yawning and swallowing. The long duration of neuralgia for five years was doubtless due to the slow development of recurrent carcinoma in the tonsil and left ary-epiglottic fold. It was the close resemblance, indeed, to this case of recurrent malignant disease in the area supplied by the left glosso-pharyngeal nerve which suggested to me that my two first cases were

really paroxysmal glosso-pharyngeal neuralgia, and not geniculate neuralgia, which I at one time suggested.

In July 1919 I saw another case of right glosso-pharyngeal neuralgia with the same throat surgeon: he had removed an epithelioma of the right palato-glossal fold from a man *æt.* 59, who afterwards complained of pain in the tonsillar region on eating and especially on swallowing a hot drink. There was no area of sensitiveness externally, and no recurrence of growth could be seen.

In the summer of 1924 I saw a man, *æt.* 54, complaining of pain in the right side of his throat which was brought on by any excitement or any muscular exertion; walking a hundred yards down the street invariably started the pain in the right side of his throat, and he had to stop and press his hand into the right side of his neck to get relief. Swallowing produced no pain, nor was there any tender area externally. In two months since the neuralgic spasms started he lost twenty-two pounds in weight, and though no neoplasm could be detected in the throat or larynx or elsewhere I was suspicious of this origin for his pain.

Very little has hitherto been published upon the subject of glosso-pharyngeal neuralgia, and beyond my own description [1] I know of only two references in the literature. In 1920 [2] Sicard and Robineau described three cases of "Algie vélo-pharyngée essentielle," which they had observed during the War; in all three cases the patients complained of spasmodic pain referred uni-laterally to the soft palate, tonsil and pharynx, lasting for years, and resembling trigeminal neuralgia in character. In all three cases the surgeon divided the glosso-pharyngeal nerve in the neck, and also the pharyngeal branches of the vagus, and ablated the superior sympathetic ganglion, the cure of the pain having lasted for four years in one case and for three years in another.

[1] *Brit. Med. Journ.*, November 26th, 1921.
[2] *Rev. Neurol.*, Vol. 36, p. 256.

Doyle,[1] of the Mayo Clinic at Rochester, Minn., in 1923 published details of four cases.

His first case, a man of 61, had had attacks of paroxysmal pain in the region of the right ear, with excessive tenderness of the right auricle for the past seventeen years, with occasional intervals, once of three years, after the tonsils were removed. The pain originally started when drinking, and this movement and eating and talking were most liable to bring on the paroxysm. At first the case was mistaken for trigeminal neuralgia major, and the sensory root of the fifth nerve was avulsed, without influencing the pain, except that it ceased for two weeks. Then the glosso-pharyngeal nerve was avulsed, and the pharyngeal branch of the vagus cut, which cured the pain. Doyle relates three other cases with paroxysmal neuralgia of similar distribution, in which swallowing especially would start the pain. One of them felt as though choking during the attacks, and another always had a hacking cough during the paroxysms. These symptoms referred to the throat recall my first case in which the patient used to arrest the painful spasms by repeatedly clearing his throat.

To sum up, glosso-pharyngeal neuralgia resembles trigeminal neuralgia major precisely in the severity and suddenness of onset of the attacks, in their brevity, and duration over a number of years. It differs from the facial neuralgia in the distribution of the pain, starting in the lateral region of the throat or tonsil, or around the ear, spreading in front of and behind the ear and to the back of the lower jaw. Repeated coughing or clearing the throat during the pain is a suggestive symptom.

Sometimes the auricle is excessively tender and sensitive. Unlike trigeminal neuralgia, glosso-pharyngeal neuralgia does not appear to have "trigger zones," though the paroxysms are started by certain movements, especially swallowing and yawning, eating and talking. Avulsion of the glosso-pharyngeal nerve as high in the neck as

[1] "A Study of Four Cases of Glosso-pharyngeal Neuralgia," *Arch. Neur. and Psych.*, Chicago, 1923, Vol. 9, p. 34.

possible is the indicated treatment, but it seems as yet uncertain whether the vagus has any share in the production of this neuralgia, and whether section of the pharyngeal branch of the vagus is also necessary, though this was done in Sicard and Robineau's three cases, and in one case of Doyle's. It is possible that alcohol injection of the glosso-pharyngeal nerve may prove a satisfactory treatment, as in my third case.

The case described in Chapter X as Auricular Neuralgia is difficult to place in terms of nervous lesion. His only trouble was in washing the ear, and of late years he let it alone, to which the appearance with the accumulated dirt on it was witness. This symptom of auricular hyperæsthesia was also present in my first two cases with glosso-pharyngeal tic, but this man had no difficulty in swallowing, and there was no pain in the throat, so glosso-pharyngeal neuralgia can probably be excluded. The vaso-motor spasm accompanying the neuralgic attacks is unlike anything I have seen in geniculate neuralgia, and it resembles the similar vaso-motor spasm that ushered in the neuralgic attacks in my case of Myoma Cutis (p. 225).

The nerve supply of the external ear is very complex. In addition to posterior auricular branches from the second and third cervical nerves, and branches from the cervical sympathetic, no fewer than four cranial nerves give branches to it: (1) the auriculo-temporal branch of the third division of the fifth nerve, (2) sensory branches from the geniculate ganglion of the facial or seventh nerve, (3) Jacobson's nerve from the glosso-pharyngeal or ninth pair, and (4) Arnold's nerve from the tenth or vagus. The vaso-motor spasm in the case above referred to is suggestive of irritation of the sympathetic, and possibly benefit would result from excision of the superior cervical ganglion.

Superior Laryngeal Neuralgia

Chronic paroxysmal neuralgia, exactly resembling trigeminal tic in its suddenness of onset, in its severity, and

in the intermissions of complete freedom from all pain, may be referred to the side of the neck, shooting up to the ear. I have seen one such case in a lady of 73, who for ten years had been subject to intermittent attacks of this nature, the pain being of intense violence, and always commencing in the same spot, which was referred to the middle of the right posterior edge of the thyroid cartilage. The pain was not felt in the fauces, there was no hacking cough during the attack, and the paroxysms were not brought on by eating, yawning or opening the mouth, though talking much might start them. The site of the pain and its general characters appear to differentiate it from glosso-pharyngeal tic, and the invariable site of origin of the neuralgia at the side of the thyroid cartilage is suggestive that it may be a *superior laryngeal* neuralgia. Injection at the point of entry of the internal branch where it pierces the thyro-hyoid membrane was therefore done with 5 minims of 90 per cent. alcohol.

Superior laryngeal neuralgia is not infrequently a result of *tuberculous ulceration* of the larynx and epiglottis. The pain is quite unlike paroxysmal tic, but chronic discomfort is present, with exacerbations of pain on swallowing, due to the friction of the food against the diseased epiglottis. Great relief may be obtained by alcohol injection of the internal branch of the nerve at the point where it pierces the thyro-hyoid membrane.

Optic Nerves

The optic nerve, though a sensory nerve, does not carry painful sensations, the afferent impressions travelling along it being limited apparently to light, colour and the pupil reflex. Yet some retinæ are more sensitive than others to certain qualities and intensities of light.

When exposed to the direct rays of brilliant sunlight, as may occur when watching an eclipse of the sun with insufficient protection in the form of smoked glass, the brilliant image of the sun focussed on the macula may irretrievably damage the sight, and a permanent central scotoma will result. In less degree a brilliant electric light, or the light from a molten steel or glass furnace, may impair vision if the eyes are insufficiently protected, and various kinds of coloured spectacles are used for this purpose. Some persons find electric light very fatiguing and irritating to the eyes, though the more yellow light of lamplight or candle does not irritate them; others again find the light of the tungsten filament lamps more irritating than carbon filaments, possibly because of the greater intensity of the illumination, and I have known simple optic atrophy develop in a watchmaker who was constantly doing fine work close to a lamp of this type. The stimulant and irritating qualities of light appear to vary largely with the amount of ultra-violet rays, and bright sunlight in high altitudes where the light is reflected from the snow and where the ultra-violet ray percentage is high is well known for its irritating effect upon the eye and in producing snow blindness. Intense photophobia and conjunctivitis are thus produced, and it may be necessary for the patient to remain in a dark room and to wear glasses for varying periods. Another curious physiological reflex of sudden bright light upon the eyes is to cause sneezing in some persons.

Optic Nerve.– The second cranial nerve or optic nerve, like the olfactory nerve, is developed as an outgrowth of the brain tissue, whose nutrient cells lie in the termination of the nerve fibres in the ganglion cells of the retina. On

their way from the brain to the retinæ the optic nerves decussate. In man, and in the apes and monkeys, and in some of the higher mammals, especially the carnivora, whose vision is stereoscopic as well as binocular, the decussation of the optic nerves is partial, about three-fifths of the fibres crossing over to the opposite side. These decussating fibres come from the inner halves of the two retinæ, which receive visual impressions from the temporal fields, which are considerably larger than the nasal fields. The site of this decussation is known as the chiasma from its resemblance to the shape of the Greek letter "χ." Practically total in herbaceous animals with panoramic vision, like the rabbit, the decussation of the optic nerves at the chiasma is total in all birds and fishes, even in those which have a high degree of binocular vision, such as owls.[1]

In man, if the nerve be severed behind the eyeball, as by a stab or gunshot wound, total blindness of that eye will result, and later on simple optic atrophy will develop, the disc as seen by the ophthalmoscope appearing pearly white. The pupil reaction to light will be completely absent to direct stimulation, as will also be the consensual reflex in the other eye, though if the sound eye be exposed to light both pupils will react practically equally.

Damage occurring at the chiasma will produce varying results, according to the exact site of the injury or point of pressure. When the latter is median, as it is likely to be when the chiasma is pressed upwards and forwards by a pituitary or infundibular tumour, the decussating fibres are those which are likely to suffer most, and defects in the visual fields will appear, producing bi-temporal hemianopia. In the early stages of this condition the defect will be found in the upper temporal field of one eye only, later involving the lower temporal, and finally the upper nasal, which is the last quadrant of vision to suffer. Not infrequently in the case of an eye apparently blind, while the optic disc appears white and atrophic, the flashing of a torch may evoke some visual response in the upper

[1] Harris, *Brain*, 1904, Vol. 27, p. 107.

Optic Nerves

The optic nerve, though a sensory nerve, does not carry painful sensations, the afferent impressions travelling along it being limited apparently to light, colour and the pupil reflex. Yet some retinæ are more sensitive than others to certain qualities and intensities of light.

When exposed to the direct rays of brilliant sunlight, as may occur when watching an eclipse of the sun with insufficient protection in the form of smoked glass, the brilliant image of the sun focussed on the macula may irretrievably damage the sight, and a permanent central scotoma will result. In less degree a brilliant electric light, or the light from a molten steel or glass furnace, may impair vision if the eyes are insufficiently protected, and various kinds of coloured spectacles are used for this purpose. Some persons find electric light very fatiguing and irritating to the eyes, though the more yellow light of lamplight or candle does not irritate them; others again find the light of the tungsten filament lamps more irritating than carbon filaments, possibly because of the greater intensity of the illumination, and I have known simple optic atrophy develop in a watchmaker who was constantly doing fine work close to a lamp of this type. The stimulant and irritating qualities of light appear to vary largely with the amount of ultra-violet rays, and bright sunlight in high altitudes where the light is reflected from the snow and where the ultra-violet ray percentage is high is well known for its irritating effect upon the eye and in producing snow blindness. Intense photophobia and conjunctivitis are thus produced, and it may be necessary for the patient to remain in a dark room and to wear glasses for varying periods. Another curious physiological reflex of sudden bright light upon the eyes is to cause sneezing in some persons.

Optic Nerve. The second cranial nerve or optic nerve, like the olfactory nerve, is developed as an outgrowth of the brain tissue, whose nutrient cells lie in the termination of the nerve fibres in the ganglion cells of the retina. On

their way from the brain to the retinæ the optic nerves decussate. In man, and in the apes and monkeys, and in some of the higher mammals, especially the carnivora, whose vision is stereoscopic as well as binocular, the decussation of the optic nerves is partial, about three-fifths of the fibres crossing over to the opposite side. These decussating fibres come from the inner halves of the two retinæ, which receive visual impressions from the temporal fields, which are considerably larger than the nasal fields. The site of this decussation is known as the chiasma from its resemblance to the shape of the Greek letter "χ." Practically total in herbaceous animals with panoramic vision, like the rabbit, the decussation of the optic nerves at the chiasma is total in all birds and fishes, even in those which have a high degree of binocular vision, such as owls.[1]

In man, if the nerve be severed behind the eyeball, as by a stab or gunshot wound, total blindness of that eye will result, and later on simple optic atrophy will develop, the disc as seen by the ophthalmoscope appearing pearly white. The pupil reaction to light will be completely absent to direct stimulation, as will also be the consensual reflex in the other eye, though if the sound eye be exposed to light both pupils will react practically equally.

Damage occurring at the chiasma will produce varying results, according to the exact site of the injury or point of pressure. When the latter is median, as it is likely to be when the chiasma is pressed upwards and forwards by a pituitary or infundibular tumour, the decussating fibres are those which are likely to suffer most, and defects in the visual fields will appear, producing bi-temporal hemianopia. In the early stages of this condition the defect will be found in the upper temporal field of one eye only, later involving the lower temporal, and finally the upper nasal, which is the last quadrant of vision to suffer. Not infrequently in the case of an eye apparently blind, while the optic disc appears white and atrophic, the flashing of a torch may evoke some visual response in the upper

[1] Harris, *Brain*, 1904, Vol. 27, p. 107.

nasal field. Occasionally complete temporal hemianopia may be found in one eye before the visual field of the other shows any defect. In March 1925 I had under treatment a young woman with acromegaly whose right eye was totally blind with optic atrophy, though five years before the right field showed well-marked temporal hemianopia, but the left field was normal.

When last seen the left eye showed commencing temporal hemianopia, the upper quadrant being blind to a small object. Such cases indicate that the upward pressure of an infundibular or pituitary tumour stretches the optic nerves at their junction with the front of the chiasma, and pressing unevenly against the inner side of one optic nerve may cause temporal hemianopia of one eye before the visual field of the other eye is affected.

Besides pituitary tumours, bi-temporal hemianopia may be produced by downward pressure of a distended ventricle in hydrocephalus, and fracture of the base may damage the middle of the chiasma, producing the same type of blinker fields. Tumours behind the chiasma may damage one optic tract, in which case homonymous hemianopia will result, the opposite fields of vision for each eye being blind; that is to say, a lesion of the left optic tract will produce blindness of the right visual fields of vision, namely, the nasal field of the left eye and the temporal field of the right eye. Each optic tract therefore contains some optic fibres which have decussated from the nasal half of the retina of the opposite eye, and also a smaller number of uncrossed fibres from the temporal half of the retina of the eye on the same side. When the damage to the nerve tract is complete, it may be possible to demonstrate Wernicke's sign, or hemianopic pupil. This is a difficult test to demonstrate, but a narrow pencil of light with parallel rays focussed upon the pupil only will probably produce no sensation of light or contraction of the pupil when thrown upon the blind half of the retina, while the pupil will react normally when the light is directed upon the seeing half of the retina.

This hemianopic pupil sign depends upon a semi-decussation of the pupil reflex fibres at the chiasma of the same type as the visual fibres.

These pupil reflex fibres run in the optic tract and leave it by the anterior brachium to reach the anterior corpus quadrigeminum. The visual fibres pass on further backwards to end in the external geniculate body, a small ganglion of nerve cells lying on the crus, and from thence new visual fibres arise and run backwards as the optic radiations to terminate in the neighbourhood of the calcarine fissure on the inner surface of the occipital lobe. Lesions of the visual fibres behind the point at which the pupil reflex fibres leave the tract by the anterior brachium will produce hemianopia but not hemianopic pupil.

Bi-nasal hemianopia is an extreme rarity, as it necessitates symmetrical damage to the outer, or non-decussating fibres on each side of the chiasma. The condition had been described as occurring in tabes dorsalis and also from the pressure of calcified carotid arteries.

Tumours at the base in the median line just in front of the chiasma may simulate pituitary tumours very closely in respect of the mental condition, increasing obesity and feebleness and the general condition of increasing blindness, but investigation will show that the blindness begins as central scotomata, and not as temporal hemianopia. Small central scotomata have been described as the commencing feature in the blinker fields of pituitary tumour, but the scotoma, though central, is really on one side only of the middle line and never advances to a large size without the temporal blindness becoming pronounced. In tumours which press upon the optic nerves in front of the chiasma, the central scotomata will increase in size practically to blindness, but peripheral vision in both temporal fields and nasal fields will be preserved as long as any vision remains. Such tumours may occur in the median line above the roof of the orbits originating from the membranes as endotheliomata, pressing upwards on the under surface of the frontal lobes and backwards on

the optic nerves between the chiasma and the optic foramen. Growths of a malignant nature also occur in the body of the sphenoid itself and are liable to cause progressive blindness first in one eye and then in the other by pressure upon the optic nerve as it passes through the optic foramen. I have known such blindness come on suddenly in one eye as the initial symptom. The pupil reflex to light will be lost to direct stimulation, but the consensual pupil reflex will be preserved so long as vision remains in the other eye. In other cases gradual strangling of the optic nerve takes place, causing central scotoma, increasing to total blindness.

Such growths of the sphenoid will soon cause limitation of the movement of the eyeballs, with diplopia, first by involving the branches of the third, fourth and sixth nerves, and subsequently causing proptosis owing to mechanical pressure of the eyeballs forwards. The clinical syndrome will then present itself as bilateral proptosis, with blindness, fixation of both eyeballs, ptosis and fixed semi-dilated pupils, headaches and increasing somnolence.

After its exit from the cranium by the optic foramen the optic nerve may be damaged by an alcohol injection for the second division of the fifth nerve, if done unskilfully. When the needle is passed in front of the coronoid process through the pterygo-maxillary fissure at an angle of 45° upwards, it should reach the foramen rotundum at the depth of about a quarter of an inch after the point has passed the external pterygoid plate, or at a total depth from the cheek varying from two to two and a half inches. If the needle be passed onwards to a greater depth in this same direction, it will be likely to hit the optic nerve at the depth of about one-third of an inch more than is required to hit the superior maxillary division of the fifth. It is therefore wise always to take the depth of the external pterygoid plate as a guide when doing this injection, and if that is done no danger to the optic nerve should ever arise.

Naso-pharyngeal tumours and growths originating in

the maxilla may also involve the optic nerve and produce blindness. Within the orbit the nerve may be involved in inflammatory changes, the result of orbital abscess, such as may result from a perforating wound or from septic absorption due to carious teeth. The eyeball will be pushed forwards and its movements limited, with partial ptosis. At first vision may not be interfered with, but in extreme cases when the proptosis reaches as much as three-quarters of an inch the optic nerve may be so stretched or involved in the inflammatory process that vision is completely lost. It is important therefore to recognise such retro-ocular abscess early so that it may be properly evacuated and drained, and vision preserved unimpaired.

Proptosis, usually bi-lateral, may be so extreme in some cases with rapidly increasing symptoms of Graves's disease that the eyeballs may be actually dislocated forwards, and sight destroyed by sloughing of the cornea and panophthalmitis.

Retro-bulbar or retro-ocular neuritis may affect the optic nerve within the orbit from a variety of causes. Local inflammation from sepsis and pressure from growths have already been referred to; in addition to these an important and common cause is a patch of disseminated sclerosis invading the nerve trunk. In this condition the eyesight may be rapidly or even suddenly lost, or gradually impaired for a time, improvement usually taking place in from a month to six weeks. Such sudden blindness occurring in a young woman who shows perhaps signs of neurotic temperament may easily be mistaken for hysterical amblyopia, a diagnosis which is apparently confirmed by the improvement which occurs in conformity with the optimistic diagnosis of the too-careless practitioner. In the amblyopic stage, however, it is quite easy to distinguish such blindness from hysteria, as the pupil reaction to light will be impaired in proportion to the loss of vision, and many tests can be devised, such as the prism test, base upwards, to show that the amblyopia is real and not functional.

With a strong prism placed in front of the good eye, base upwards, and with both eyes open, in cases of hysterical amblyopia, or in malingering, vertical diplopia will be produced, one set of test types being seen above the other, the lower image belonging to the "good" eye. With a little management and encouragement, such as assuring the patient that the effect of the prism is to cause the good eye to see double, the visual acuity of each eye can be tested and compared, without the patient being aware that the "bad" eye is also being tested.

In the amblyopia of retro-bulbar neuritis or other organic disease, the prism will cause diplopia if the other eye has any vision, but only one image will be distinct. In using this test it should be remembered that the image is deflected in the direction of the apex of the prism.

Careful examination of the visual field in retro-bulbar neuritis will always show that the blindness is really due to a large central scotoma, so that objects directly looked at, such as the face of a clock, will not be seen, though surrounding objects may be fairly distinct. Ophthalmoscopic examination will generally demonstrate an actual neuritic condition of the optic disc, indeed the swelling may sometimes be so pronounced as to simulate in slight degree the choked disc of cerebral tumour. As a rule the swelling of the disc does not exceed between one to two diopters, and subsequently pallor of the disc will supervene, the temporal side of the disc showing definite signs of atrophy, and in severe cases the whole optic disc will be pearly white. In rare cases vision may be lost to the degree of perception of light only, but total blindness is practically unknown in disseminated sclerosis, though it is common enough in the primary optic atrophy of tabes dorsalis.

In addition to disseminated sclerosis, retro-bulbar neuritis may be due to a variety of toxic causes, of which tobacco amblyopia is fairly well known. I have also seen it result from sudden shock from a gas explosion and also from a

vivid flash of lightning; in each case blindness was immediate and succeeded by simple optic atrophy.

Central scotoma has been described as occurring in the atrophy of tabes dorsalis, but usually the visual fields are concentrically narrowed with the diminishing acuity of vision. Unlike disseminated sclerosis, the primary atrophy of tabes dorsalis constantly proceeds to total blindness, the lesion here being parenchymatous degeneration of the nerve fibres, but in disseminated sclerosis the lesion is primarily inflammatory in the fibrous tissues surrounding the nerve bundles, the atrophy being subsequently due to the contracting scar tissue destroying the axis cylinders. Primary optic atrophy is met with occasionally in other chronic diseases such as Sanger Brown's type of cerebellar ataxy, and it may occur in families of the type known as Leber's familial atrophy.

I have already referred to primary optic atrophy occurring as an occupation neuritis, from constant exposure to strong electric light.

Secondary or post-neuritic atrophy of the optic disc, with partial or complete blindness, is a common sequel of the papillœdema resulting from raised intracranial pressure, as in cerebral tumour; the visual fields in these cases will usually be contracted without central scotomata.

Ophthalmoscopic examination will often, though not invariably, enable such secondary atrophies to be distinguished from a primary atrophy met with in disseminated sclerosis or tabes.

Atrophy of the disc may result from local damage within the eyeball such as prolonged raising of the intra-ocular tension in glaucoma, and it is also a secondary result of retinitis and choroiditis. Embolism of the central artery of the retina will also cause sudden amblyopia, with succeeding optic atrophy, in which case extremely narrowed branches of the central artery will usually be diagnostic; a somewhat similar condition, however, may arise in thrombosis of the central artery secondary to vasomotor spasm after a large dose of quinine, known as quinine amblyopia. Some-

times quinine amblyopia recovers, the arterial spasm relaxing, but it is a very dangerous condition and may result in total permanent blindness.

Primary optic atrophy was formerly frequently produced as a result of prolonged administration of organic arsenical compounds such as atoxyl, which was largely used for sleeping sickness and trypanosomiasis in East Africa, and also in the treatment of lymphadenoma and other grave anæmias.

No danger of optic atrophy is likely to arise if no more than a total of twelve grains of the arsenical compound has been given. The arsenobenzol compounds used in the treatment of syphilitic diseases do not appear to have the same toxic effect upon the optic nerve fibres.

Third, Fourth and Sixth Cranial Nerves

The movements of the eyeball are performed by six muscles inserted into its outer fibrous coat, the sclerotic. Of these four receive their nerve supply from the third or oculo-motor nerve, namely the superior and inferior rectus, the internal rectus and the inferior oblique. The remaining two muscles are each supplied by a separate cranial nerve, the superior oblique being supplied by the fourth nerve and the external rectus by the sixth nerve.

Though in infancy the movements of the eyeballs are not entirely co-ordinated, it being a quite common thing for squints to be seen in children with normal eyes in the first year of life, yet later on, as stereoscopic binocular vision becomes developed, and the children learn to focus clearly, the movements of the eyes become completely synchronised.

The normal position of the eyes at complete rest is one of distinct divergence, as can be seen in coma or during sleep. The co-ordination of the movements of the eyes is a complex process in which many portions of the brain take part, which need not be gone into here in detail.

Third Nerve.—The third nerve originates from a mass of grey matter beneath the floor of the aqueduct in the

upper portion of the pons and immediately beneath the anterior corpora quadrigemina. This nucleus really consists of several masses of grey matter which are the nuclear origin for the groups of fibres concerned with the different functions of the third nerve. Accommodation by contraction of the ciliary muscle within the eye, and contraction of the pupil by the sphincter iridis, are served by two bundles which arise from the most anterior portion of the third nucleus. Behind these come the nuclear origin for the internal rectus, the superior and inferior recti, the inferior oblique, and the levator palpebræ. Disease of the anterior portion of the nucleus will produce a condition called internal ophthalmoplegia, or paralysis of accommodation and of contraction of the iris. Partial internal ophthalmoplegia is a common temporary sequel of diphtheria, loss of power of accommodation being due to diphtheritic neuritis affecting the ciliary muscle, while at the same time there is usually some sluggishness of the pupil reaction to light. Total internal ophthalmoplegia, the pupil being dilated and immovable, with complete loss of power of accommodation, is generally nuclear in origin and a sign of neurosyphilis. Disease, however, affecting the ciliary ganglion in the orbit may also produce the same condition.

Lethargic encephalitis frequently affects the third nerve nuclei, sometimes causing double third nerve paralysis, bilateral ptosis and divergent strabismus.

It is this drooping of the lids which contributes to the sleepy appearance of the patients, and not all sufferers from this disease are lethargic and sleepy, for insomnia is sometimes a prominent symptom. Many of the subjects of lethargic encephalitis complain of diplopia, and nystagmus is common, while ptosis may be completely absent. The pupils are, however, usually affected, being unequal and reacting badly to light. This condition may remain as a permanent sequel of the disease even though the diplopia due to partial third, fourth or sixth nerve involvement may have disappeared.

External ophthalmoplegia is a term referring to a

condition of partial or complete immobility of the eyeball due to paralysis of the external muscles moving the eyeball. This may be due either to nuclear disease or to lesions of the nerve branches, as in multiple neuritis. External ophthalmoplegia is sometimes seen in exophthalmic goitre without sufficient proptosis to explain the fixation of the globes, and it may also be pronounced in myasthenia, in which disease it is apparently the muscles themselves that are affected, and not their nerve supply. Disease affecting the anterior corpora quadrigemina, such as vascular lesions, or growths, tumours of the pineal body which lies in the median line just in front, or tumours growing in the third ventricle are liable to cause varying degrees of ophthalmoplegia, the pupils particularly being paralysed in dilatation by tumours in this region.

The third nucleus may suffer from chronic degeneration of its nerve cells as a late sequel of syphilis, or it may be damaged by neighbouring hæmorrhage, or by softening from thrombosis, or by growth. The nerve fibres arising from the various portions of the third nucleus are gathered together into a bundle, which passes ventrally through the anterior portion of the pons near the middle line to leave the brain stem in the depression between the crus and the pons; thence they pass forwards at the base of the brain along the under side of the crus to reach the outer wall of the cavernous sinus, being enclosed in a sheath of dura mater. Passing forwards, the nerve divides into branches which leave the cranial cavity by the sphenoidal fissure, to enter the back of the orbit and to supply the various muscles enumerated above. Within the orbit and behind the eye is a small ganglion of nerve cells, the ciliary ganglion, which receives its motor root from the branch of the third nerve to the inferior oblique. From the ciliary ganglion the motor fibres for the ciliary muscle and sphincter of the iris pass to the eyeball.

After leaving the pons the third nerve is therefore exposed to damage from many causes at the base of the brain, such as meningitis, fractured base, hæmorrhage and

growth. Complete third nerve paralysis is a not uncommon result of syphilis, due to gummatous neuritis, and it is also frequently met with in tabes dorsalis and in general paralysis of the insane.

Total third nerve paralysis is easily recognisable by the presence of complete ptosis and external divergence of the eyeball, while the eye cannot be rotated upwards and scarcely at all downwards, though, by means of the superior oblique, inward rotatory and downward movements may be observed. Owing to the unopposed power of the external rectus the eyeball is turned completely outwards, so that the iris reaches the external canthus. In addition to these signs of external paralysis the pupil will be semi-dilated and will not react to light or to attempts at convergence, and the power of accommodation will be entirely lost. Such total third nerve paralysis will be rare in the absence of syphilis or tabes.

Slighter degrees of weakness are common, not only in syphilis, but as the result of acute encephalitis, fractured base, meningitis, growth, diphtheritic or alcoholic neuritis, in myasthenia or as a distant pressure symptom in cerebral tumour. These will vary from almost complete paralysis of the nerve down to slight drooping of the upper eyelid, with diplopia, but showing no obvious weakness of an external muscle. Slight degrees of third nerve paralysis may also be produced occasionally as a temporary result of alcohol injection either into the Gasserian ganglion, or of the second division of the fifth nerve at the foramen rotundum. In the former case some of the alcohol leaks through into the lateral recess, where either the third or the sixth nerve may be involved.

Ophthalmoplegic Migraine

This disease is entirely distinct from ocular migraine or migraine ophtalmique, though it resembles the latter in periodicity of attack, with violent pain on one side of the head, which may be accompanied by vomiting. The pain in ophthalmoplegic migraine is often referred to the back

of the eye, spreading over the brow and temple on that side, and resembles the local pain in the form of migraine that I have described as "Ciliary Neuralgia." The attacks last much longer than those of migraine, from three or four days to as long as a month, and are accompanied by the development of third nerve paralysis, always on the same side. Vomiting is common during the earlier part of the attack, and drowsiness and torpor, even convulsions, may occur. I have seen the condition mistaken for lethargic encephalitis. The frequency of the attacks varies greatly from intervals of weeks to months or even years. Occasionally the attacks become more and more frequent, the third nerve becoming ultimately completely paralysed, and the pain in the orbit may become so intense that life is a misery. In one of my cases the patient got no relief from the intense pain until in desperation he insisted on the eyeball being removed, the attacks then ceasing. In another man, *æt.* 30, repeated attacks had left the third nerve totally paralysed with complete ptosis between the attacks, and at the commencement of each bout of violent pain the upper eyelid would flicker up and down, suggestive of an irritative process affecting the third nerve. The disease may commence with sudden violent pain behind the eyeball, and ptosis and third nerve palsy may become nearly complete within two or three days, though after the attack is over and the pain has disappeared the third nerve paralysis gradually recovers, at first almost completely, though with each subsequent attack the recovery of the ocular palsy is less complete, until finally no improvement in the ptosis or external strabismus is seen in the intervals between attacks. I have seen one similar case in which the sixth cranial nerve was intermittently paralysed in place of the third nerve, though in other respects the case resembled opththalmoplegic migraine.

Local trauma to the eye itself may determine the onset of the attacks, and the third nerve paresis may be partial. In the following case the recurrent paresis was limited

to complete internal ophthalmoplegia and partial ptosis, without any diplopia or weakness of any external ocular muscle. A girl, *æt.* 17, had some salt thrown in her right eye, causing considerable pain and irritation, with some conjunctivitis and pain over the right eyebrow. Two days later she noticed drooping of the eyelid and blurred vision of the right eye for near objects, though distant vision was unaffected. During the next two years she had eleven similar attacks. Each was preceded by symptoms of conjunctivitis and pain over the right eyebrow, and followed on the next day by partial ptosis and a semi-dilated fixed pupil, not reacting to light, with loss of accommodation. The attacks always cleared up entirely in about six days. The limitation of the total paresis to the pupil and the ciliary muscle was suggestive of loss of function of the ciliary ganglion.

The *pathology* of this disease is very obscure. In a few cases exudation has been described as being found on the third nerve post mortem. In others injury from a fall during childhood has damaged the third nerve at the base of the brain. The reference of the intense pain to the back of the eyeball, and the fact that injury to the eye may originate the disease, and that in one patient the attacks of pain ceased after enucleation of the eye, are suggestive that in some of the cases at least the lesion may occur as far forward as the sphenoidal fissure or even within the orbit.

In *myasthenia gravis,* ptosis and diplopia from weakness of ocular muscles, or even a complete fixation of the eyeballs from external ophthalmoplegia, may suggest partial third nerve paralysis, though no lesion of the nerve has ever been discovered, and the morbid process appears to be limited to the muscles themselves. The feature of the myasthenia is the variability of its symptoms, and particularly the effect of fatigue in augmenting the paralysis, which may disappear after a rest for an hour or more. In this disease neither the pupil reflex nor the ciliary muscle will be interfered with, the ocular weakness being limited to the external muscles of the eye.

Rheumatic neuritis may attack branches of the third nerve, causing diplopia for a few weeks, usually without pain. When this form of neuritis attacks the ciliary ganglion there is likely to be intense pain in the eyeball and orbit, owing to the involvement of the ciliary nerves from the ophthalmic branch of the fifth nerve. The nerve to the inferior oblique is also likely to be paralysed at the same time, causing homonymous diplopia, and the pupil reaction and the ciliary muscle for accommodation will also be weakened. Such cases must be treated by the administration of salicin or salicylates, by fomentations over the eyeball and by keeping the eye covered up, and the pain may be sufficiently intense to necessitate the use of morphine.

Within the cranium lesions of the third nucleus or of the third nerve at the base of the brain will be likely to involve the pyramidal tract as it passes from the ventral portion of the crus into the pons, thereby producing hemiplegia on the opposite side, a form of crossed paralysis, the third nerve on one side being affected, with hemiplegia of the face, arm and leg on the opposite side. I have seen such a condition as a permanent result from a severe fall in a child at the age of three. When adult the patient had almost complete left third nerve paralysis, with rigidity and extreme athetosis of the right arm, the leg being less affected.

The Fourth Nerve, or *nervus patheticus*, arises from a nucleus which is almost a continuation posteriorly of the third nucleus. Instead of running ventrally, however, like the third nerve, the fibres of the fourth nerve turn dorsally around the aqueduct and decussate in the middle line of the velum interpositum on the roof of the fourth ventricle. Thence the fibres wind round the crus to reach the outer wall of the cavernous sinus, thence passing through the sphenoidal fissure to supply one muscle only, the superior oblique. This muscle has a curious mode of insertion into the eyeball, its tendon running through a pulley-shaped groove near the inner canthus of the eye,

whence it runs back over the globe to be inserted into the posterior portion of the eyeball. The action of this muscle is to turn the eye slightly outwards and downwards, with some rotation inwards.

The diplopia produced when this muscle is paralysed will therefore be homonymous, with the false image slightly depressed and its upper margin tilted inwards. Although the action of the muscle alone is to turn the eye outwards, yet in this movement its tendon does not run over the greatest equator of the eyeball, and hence its leverage will not be so great as when the eye is turned inwards by action of the internal rectus. In this latter position the diplopia produced by superior oblique palsy may be more noticeable to the patient than when the eye is turned outwards.

The method for determining the paralysed muscle in cases of diplopia is as follows: (1) Is the diplopia crossed or homonymous? a question which is easily answered by closing and reopening one eye two or three times while the patient fixes his attention on a given object. In homonymous diplopia the image belonging to each eye is on the same side as that eye. Homonymous diplopia indicates a convergent strabismus, that is to say, paresis of one of the muscles which turns one of the eyes outwards, either the external rectus or the superior or inferior oblique. This test, therefore, narrows down the inquiry from twelve possible muscles to six. (2) Does the diplopia become more marked when the patient turns his eyes to the right or to the left? This test will narrow down the inquiry still further from six muscles to three, as, for example, an homonymous diplopia which increases in extent when the eyes are turned to the left must mean weakness of either the left external rectus or of one of the two oblique muscles of the left eye. To take another example: a crossed diplopia which becomes more marked on looking to the right. Crossed diplopia indicates weakness of one of the six muscles which turn either eye inwards. Consequently, if the diplopia increases to the right the weak muscle must be either the left internal rectus or the

left superior or inferior rectus. (3) Study of the false images will then decide which muscle is at fault. The false image is projected in the direction of the action of the paralysed muscle. Therefore if the external rectus is paralysed the false image will be erect and outside the true image, and at the same level, whereas with the oblique muscles paresis of the superior oblique will produce a false image on the outer side of the true image and it is depressed below it and has its upper margin turned inwards. The false image due to paresis of the inferior oblique will be similarly outside the true image, but above it and with its upper margin rotated outwards.

Conversely, diplopia due to paresis of the internal, superior, or inferior rectus will be of the crossed variety, that is to say, when an object is looked at and diplopia occurs, closing the right eye will abolish the left-hand image and vice versa. When this is the case the diplopia will be most marked when the eyes are turned away from the paralysed side. The image will be erect, crossed, and on the level when due to internal rectus paresis : in paresis of the superior rectus the false image will be crossed, above the true image, and with its upper margin turned outwards, while in paresis of the inferior rectus the false image will be crossed, depressed below the true image and with its upper margin turned inwards.

In cases of slight diplopia it is often difficult for the patient to be consistent in his answers owing to the fleeting character of the false images, and it will be advisable then, if accuracy of diagnosis is desired, to test the patient in a dark room, using a lighted candle, and a piece of red glass held in front of one of the patient's eyes. The head of the patient should be steadied and kept in one position, while the eyes are made to follow the movements of the candle in nine separate positions, that is to say, straight in front, to right and to left; above, horizontally and downwards.

Isolated paralysis of one fourth nerve is not a very rare occurrence, and is apt to cause troublesome diplopia,

because it is most marked when the patient is looking downwards, as when descending the stairs or when walking on the pavement. Slight diplopia is apt to be more troublesome than extreme diplopia, because when the false images are widely separated the patient has less difficulty in suppressing the false image, but when the false image is close to or overlaps the true image, the confusion will be more noticeable. Within the brain the fourth nucleus may be damaged together with the nucleus of the third nerve, while after leaving the pons one or both fourth nerves may be damaged at the roof of the fourth ventricle by meningitis or by pressure of a pineal or cerebellar tumour. After winding round the crus it may be involved in meningitis or gumma, and on reaching the external wall of the cavernous sinus it may suffer like the third or the sixth nerve from thrombosis of the cavernous sinus, and in the orbit from rheumatic or septic inflammations.

The weakness of external deviation of either eye due to paresis of the superior oblique is often difficult to demonstrate, though on external deviation of the eye by the external rectus it may be possible to see slight rotation of the eyeball outwards from unopposed action of the inferior oblique.

Isolated paralyses of the third or fourth nerves, though common enough in syphilis and tabes dorsalis, are probably never met with in disseminated sclerosis, though diplopia is one of the commonest symptoms in this disease. Weakness of any of the movements inwards, outwards, upwards or downwards may be demonstrable in disseminated sclerosis, though analysis will rarely show it to be due to weakness of any single muscle, but rather the weakness is of co-ordination and of conjugate movement of the two eyes. That this is so, and that the lesion is a central one of the co-ordinating centres and their afferent paths, is proved by the frequent presence of nystagmus in association with diplopia in this disease. Often the type of nystagmus in disseminated sclerosis is characteristic,

there being in reality a weakness of the lateral conjugate movement of the two eyes to each side, which shows itself by defective inward movement of the one eye, with a coarse nystagmus of the other or outer eye, the quick movement of the nystagmus reaching the outer canthus, but the effort of deviation not being properly maintained the eye swings slowly inwards, only to be turned outwards rapidly again by a new effort at external deviation.

Thus, when the eyes are turned towards the left, nystagmus of this type is seen in the left eye with weakness of inward movement of the right eye, causing apparent divergent strabismus. When the eyes are turned to the right the converse condition is seen, nystagmus of the right eye with weakness of inward deviation of the left eye.

Sixth Nerve Paralysis.—The sixth nerve arises from a somewhat globular nucleus close to the junction of the medulla and pons, and beneath a slight swelling in the floor of the fourth ventricle known as the eminentia teres. This nucleus has connections with higher centres co-ordinating the ocular movements, and it not only gives off the fibres of the sixth nerve which leave the brain stem at the junction of the medulla and pons on its ventral surface, but it also sends fibres forwards through the posterior longitudinal bundle, which decussate in the neighbourhood of the third nucleus in order to join the third nerve of the opposite side, ultimately innervating the internal rectus muscle. This muscle has therefore a double nervous supply, both from the third nucleus and from the opposite sixth nucleus. The latter nucleus governs the conjugate movement of the two eyes to the same side, the left sixth nucleus by means of the left sixth nerve and the external rectus muscle deviating the left eye outwards, while through the posterior longitudinal bundle the opposite internal rectus pulls the right eye inwards. The two sixth nuclei may therefore be compared to a coachman's hands guiding a pair of horses by reins to right or left. When both sixth nuclei therefore are destroyed the eyes cannot be moved laterally at all, but can be moved upwards or downwards, and can converge inwards, through

the action of the third nuclei. This is a physical sign often seen in glioma of the pons, in children.

The sixth nerve runs a long course along the base of the brain to reach the outer wall of the cavernous sinus, and it is therefore exposed more than any other cranial nerve to the effects of indirect pressure from cerebral tumour. Paralysis therefore of one sixth nerve will be of little localising value for cerebral tumour. This nerve is also subject to damage from fractured base or from meningitis, being often affected early in tuberculous meningitis. After leaving the wall of the cavernous sinus the sixth nerve passes through the sphenoidal fissure on its way to the orbit; in this region it may suffer from rheumatic neuritis, as a rule not accompanied by pain, the diplopia lasting from three to six months. Like the other structures in the orbit it may suffer from septic infection secondary to dental caries, retro-ocular abscess, etc.

Seventh or Facial Nerve.—The seventh cranial nerve, the motor nerve of the facial muscles, once called the portio dura, was originally a nerve of the first gill slit, and it still preserves its relationship to the first gill slit or spiracle in its anatomical course around the middle ear and its exit behind and beneath it at the stylo-mastoid foramen. The developmental interdependence of these two structures is seen in certain rare cases of congenital absence of the outer ear, a condition which is associated with congenital facial paralysis of the same side owing to the non-development of the facial nerve. In these cases other teratological malformations are likely to be present.

Formerly considered as a purely motor nerve, it is now known, mainly through the researches of Ramsay Hunt, that it furnishes some sensory supply to the skin of the auditory meatus and pinna, and that troublesome neuralgias may be connected with this sensory supply (see Geniculate Neuralgia).

The facial nerve takes its origin in the medulla from a nucleus slightly posterior and ventral to the sixth nucleus; its fibres pass upwards and inwards and around the sixth nucleus, before they emerge as the facial nerve at the side

of the medulla. Facial paralysis, therefore, of infra-nuclear type is apt to be produced in lesions damaging the sixth nucleus.

Vascular lesions such as hæmorrhage or thrombosis may damage the seventh nerve in this region, and even acute degeneration of the seventh nucleus is sometimes seen in acute anterior poliomyelitis. Nuclear facial palsy may also be produced through gross lesions affecting the medulla such as glioma, tuberculoma, or syringobulbia.

After its emergence from the medulla the nerve may suffer damage from meningitis, gumma or growth, or it may be pressed upon by a cerebellar tumour or may be implicated in a fractured base. It enters the internal auditory meatus in company with the auditory nerve and with the small nerve known as the pars intermedia Wrisbergi. This latter nerve is connected with the geniculate ganglion, a small collection of nerve cells usually described as lying upon the facial nerve at the point where it bends backwards at a right angle over the promontory in the middle ear.

These nerve cells have really no connection with the motor fibres of the facial nerve, but may be looked upon as homogeneous with the Gasserian ganglion on the fifth nerve, or the petrous and jugular ganglia of the glosso-pharyngeal and vagus nerves, and comparable to a posterior spinal root ganglion. The pars intermedia Wrisbergi is thus the central prolongation of the geniculate ganglion and comparable to a posterior spinal root, while the chorda tympani, the great superficial petrosal nerve, and the sensory fibres supplying the posterior wall of the external auditory meatus and part of the back of the pinna, which are the peripheral branches of the ganglion, are homologous to peripheral sensory nerves.

It is owing to the involvement of these latter sensory fibres in lesions involving the facial nerve trunk in the region of the ear that pain accompanies many cases of Bell's palsy, preceding by a day or two and lasting for a few days after the onset of facial paralysis.

CHAPTER XVIII

FACIAL PARALYSIS. CLONIC FACIAL SPASM

Facial Paralysis or Bell's Palsy is a common disease at almost all ages. Occasionally sufferers from this complaint are very frightened of it because in their ignorance they think it is a stroke. Such peripheral facial paralysis has nothing in common with the weakness of the lower portion of the face that accompanies weakness of the arm and leg on the same side in ordinary apoplexy. Rheumatism and chill are often blamed as a common cause of Bell's palsy, and there is little doubt that direct exposure to cold may produce it. The old and popular superstition that the face will become drawn to one side if the moonlight is allowed to shine upon it is no doubt dependent upon observed cases of facial paralysis when sleeping in the open. The site of the lesion of the nerve in these cases is a matter of dispute and possibly it varies. Many cases appear to be a degenerative neuritis commencing just peripherally to the geniculate ganglion.

The nerve running here in a bony canal, congestion and swelling of the sheath of the nerve will cause pressure upon the chorda tympani and other sensory fibres in addition to the motor facial nerve, and consequently it is no matter of surprise that interference with taste sensations on the side of the tongue commonly accompany such facial paralysis. Sometimes the loss of taste is complete over the front half of the tongue, and in a few cases it will be possible to detect slight diminution in tactile sensibility of the tongue. In certain cases, where the neuritis has probably attacked the geniculate ganglion itself, a herpetic eruption may accompany the facial paralysis, the distribution of the rash being found along the posterior wall of the

external auditory meatus and even extending on to the tympanum, with sometimes a zone of herpes behind the ear at the junction of the pinna with the scalp; or, again, the herpetic eruption may be seen along the side of the tongue and even on the soft palate and back of the hard palate.

In other severe cases the facial paralysis will be complete, and the deformity great, the mouth and philtrum being drawn over to the sound side, with smoothing out of all the

Fig. 41.—Facial paralysis, or Bell's palsy, right side. The patient is endeavouring to screw up both eyes and to wrinkle the forehead.

wrinkles on the paralysed side, the cheek blown out in respiration owing to paralysis of the buccinator muscle, with inability to close the eye and liability to lachrymation. This tendency to overflow of tears is due to two causes, first, falling away of the punctum from contact with the ocular conjunctiva owing to paralysis of the orbicularis muscle in the lower lid, and consequently the non-aspiration of the tears into the lachrymal sac, and, secondly, the development of conjunctivitis owing to inability to close the eye because of the orbicularis paralysis. All the muscles supplied by the facial nerve will be equally paralysed,

including the frontalis and corrugator in the forehead, and the orbicularis oris or sphincter muscle of the lips, muscles which escape in hemiplegic paralysis of the face. The platysma muscle in the neck is also paralysed, and a small muscle in the middle ear, the stapedius, will also be paralysed, producing a symptom sometimes met with, hyperacusis. This excessive sensitiveness to noise is more evident to raucous clashing noises such as the barking of a dog or a grinding rattle than to clear musical notes. It is a symptom only rarely met with. In slighter cases the loss of voluntary power may be apparently equally marked, though there may be little or no deformity noticeable owing to the continued presence of tonic contraction of the muscles.

Such recovery of tonic contraction will be the first sign of recovery from total paralysis before any sign of voluntary power returns, and it is of the highest cosmetic value to the patient in abolishing the deformity of the features. In many cases of paralysis of moderate severity little or no voluntary power may ever return, though the tonic contraction, and later, contracture, reduce the deformity to a minimum.

Chronic discharge from the ear following perhaps scarlet fever, measles or other sources of acute middle ear abscess may in course of time produce facial paralysis through spread of the inflammatory process to the sheath of the facial nerve. In certain cases this occurs only after erosion of the bone has taken place, while in others the facial nerve may be exposed from the first, owing to a defect in the bony canal.

As a rule facial paralysis produced in this way is apt to be severe, leading either to total paralysis with severe deformity or else to considerable contracture of the paralysed muscles. Taste is almost certain to be lost on the anterior two-thirds of the tongue. Pains may accompany this type of facial paralysis, and the electrical changes in the muscles and nerve will be the same as in Bell's palsy resulting from chill or infection.

Mastoid operations for the treatment of chronic otorrhœa or acute mastoiditis are sometimes followed by facial paralysis either slight or severe. When the facial paralysis is noticeable immediately after the operation this result may be due to jarring of the nerve from concussion, as when a heavy mallet is used. In some unfortunate cases the operation is even more directly responsible for the facial paralysis by direct injury of the nerve being caused by the blow of a chisel, or being damaged by a curette. In skilled hands, however, such direct injury is now very rare.

In total paralysis the deformity of the face is permanent, and electrical testing shows at first reaction of degeneration, with gradually decreasing strength of contractions to galvanism as the weeks go by.

If after three months there is no sign or return of tonic contraction the question of operation by nerve anastomosis may be considered. For this purpose the facial nerve is exposed behind the angle of the jaw and either the hypo-glossal nerve or the spinal accessory may be used for the purpose. The facial nerve is cut across as high as possible, and the end of the peripheral portion is carefully inserted into a nick made in the side of either the hypo-glossal or spinal accessory nerve. The object of this nerve anastomosis is to allow of the down-growth of motor nerve fibres into the peripheral portion of the facial nerve. The immediate result will often be weakness of the sterno-mastoid and trapezius if the spinal accessory nerve be chosen, or of the half of the tongue when the hypo-glossal is made use of. No immediate improvement in the facial paresis is to be looked for, though after three months some signs of recovery of tone and voluntary movements may be perceptible. At first such movements of the face will accompany the movements of the head and shoulder when the spinal accessory is the auxiliary nerve, or they will be associated with the tongue movements when the hypo-glossal has been used. Eventually some differentiation of the facial movements may be possible from the function of the other two nerves.

This operation of nerve anastomosis is not to be recommended unless the facial paralysis is total and complete without any return of tonic contraction. Even then the operation is somewhat of a gamble, as there is a possibility, not only of failure to improve the facial deformity, but also of production of paralysis of the spinal accessory and hypo-glossal nerves.

Fig. 42.—Clonic facial spasm, the sequel of left facial paralysis sixteen years previously, the patient also being subject to paroxysmal trigeminal neuralgia of the left third division, which was relieved by alcohol injection, without influencing the motor spasms.

From the cosmetic point of view the appearance of facial contracture in cases of moderately severe facial paralysis is very valuable in reducing deformity, so that in many cases in which there may be practically no return of voluntary power on the paralysed side, yet the appearance of contracture of the paralysed muscles deepens the naso-labial fold and draws the mouth straight, so that at rest there may be little or no sign of paralysis of the face. Indeed this tendency to contracture is very apt to be over-

done, so that a contracture of the orbicularis palpebrarum appears to make the ocular fissure smaller, and the naso-labial fold deeper than on the sound side, so that at rest it might be possible to mistake the paralysed side with contracture for the sound side. In addition to contracture, a not infrequent sequel of moderately severe facial paralysis is *clonic spasm* of the paralysed muscles. These intermittent spasmodic contractions are usually not severe enough to cause much inconvenience to the patient, and differ thereby from the severe form of facial spasm which develops gradually without preceding facial paralysis. Rarely the clonic spasm succeeding an attack of facial paralysis may be as intense and sudden in the contractions as in the form of idiopathic clonic spasm to be described presently. I have seen one such case associated with chronic neuralgia of the face of sixteen years' duration in a woman of 72 (Fig. 42).

In the association of severe facial neuralgia on the same side as the strong clonic facial spasm, the case closely resembled the cases described by Cushing as painful tic convulsif, though in my case the strong clonic facial spasms were not synchronous with the neuralgic spasms.

The contracture and clonic spasm of muscles paralysed as a result of neuritis are peculiar to the facial nerve, and are not met with in any other form of neuritis of a peripheral nerve. It has been suggested by Ramsay Hunt that these phenomena are the result of reflex irritation of the motor facial nucleus through afferent stimuli from the geniculate ganglion.

Facial paralysis is at times truly peripheral in type, that is to say, the neuritis affects, not the trunk of the nerve close to the geniculate ganglion, but the peripheral branches in the parotid region and cheek.

Possibly this form of paralysis may be due directly to chill, but more commonly it is of the toxæmic type and associated with other signs of toxic polyneuritis. Alcoholism may be a contributory factor to the neuritis, but more commonly the multiple neuritis is not dependent upon

alcoholism, but is the result of toxæmia of unknown origin which was first brought clearly into prominence during the Great War. Ushered in by a period of malaise, furred tongue, constipation and vague general pains, with pyrexia for three or four days, this stage is followed by apparent recovery until, three or four weeks later, a recurrence of severe general pains takes place, with intense depression, muscular tenderness and signs of general multiple neuritis,

FIG. 43.—Partial facial diplegia, or bilateral paralysis of the sphincters of the lips and eyelids. The patient is endeavouring to close both eyes, and to purse up the mouth. A case of Myasthenia gravis.

hyperæsthesia and pins and needles of the extremities, with some muscular weakness of the extensors. At the same time, facial paralysis will be noticeable, commencing on one side and often involving both sides of the face. There will be no loss of taste in this type of facial paralysis, probably because the neuritis is truly peripheral and affects the nerve fibres beyond the point at which the chorda tympani nerve joins the facial nerve in the middle ear. When the facial paralysis is bi-lateral there will be no distortion of the mouth or features, and indeed the presence

of bi-lateral facial paralysis is often overlooked. There will be weakness or inability to close the eyelids, and the movements of the lips and angle of the mouth in smiling and showing the teeth will be impossible.

The electrical reactions will show slight diminution to the faradic current, but full reaction of degeneration is not likely to occur, and the facial paralysis usually begins to improve after two or three weeks and complete recovery is the rule. The treatment of this condition will follow the usual lines in polyneuritis : rest in bed, warmth, hypnotics when necessary, with massage and general faradism, and the administration of strychnine as soon as the hyperæsthesia and other sensory symptoms permit.

Bi-lateral facial paralysis may occur alone as a double Bell's palsy, though usually there is an interval either of months or years between the affection of the two sides. Middle ear disease may also be the cause of bi-lateral facial paralysis, particularly as a sequel of scarlet fever or measles.

A special form of bi-lateral facial paralysis accompanied by bi-lateral total nerve deafness is sometimes met with as a result of secondary syphilis, being an inflammatory neuritis of both seventh and eighth nerves. It will be distinguished from the form due to middle ear disease by the absence of any history of otorrhœa. Nerve deafness is total in this condition, even a brass band being inaudible, though the vibrations of a drum will be perceptible to the patient. In this form of bi-lateral facial paralysis the nerves are totally destroyed, and reaction of degeneration is present for the first few months, though ultimately no electrical reactions can be obtained. I was once able to use this test to prove a woman to be a malingerer who had obtained admission to hospital under false pretences, having been found clinging to the railings and telling the police who brought her in that she had had a stroke and lost her hearing. Testing her facial muscles with faradism and galvanism proved that the paralysis was at least six months old.

Bi-lateral facial paralysis may also be due to other forms of basal meningitis or chronic tumours at the base of the brain, such as bi-lateral acoustic fibro-sarcomata which grow from the sheath of the auditory nerves close to the internal auditory meatus, and may involve the facial nerves.

Treatment of Facial Paralysis.—For the first week the best treatment is to apply fomentations over the ear and parotid region, precautions being taken against any chill or exposure to cold. Otorrhœa and mastoiditis, of course, require special treatment, but the ordinary Bell's palsy or so-called rheumatic facial neuritis is best treated at first by the direct application of warmth; indeed, with energetic and frequently renewed poultices and fomentations I am convinced, if they are applied early enough and with persistency for some days, that the severity of the facial paralysis may often be considerably lessened and the resultant deformity diminished, while less severe cases may clear up within a week or ten days.

As soon as any pain associated with facial paralysis has disappeared, electrical treatment in the form of labile galvanism should be commenced, the negative electrode being held beneath the mastoid, and the positive electrode, or that attached to the carbon element of a box battery, should be stroked over the paralysed muscles for about a quarter of an hour twice a day. The anode or positive electrode should be used for application to the face because it is less painful than the cathode. Weak currents should be employed, not more than three milliamperes being usually necessary. The strength of contractions to this current will depend upon the degree of hyperexcitability of the muscles, and in some of the slighter cases of neuritis, although voluntary power may be completely lost, no galvanic hyperexcitability may appear, and even the faradic reactions may persist. The prognosis in these cases is very good and practically complete recovery will ensue.

Clonic Facial Spasm

Clonic facial spasm as a sequel to partial recovery from facial paralysis has already been described (see page 364).

Non-painful facial tic, or clonic facial spasm affecting the whole musculature supplied by the facial nerve on one side, is to be distinguished from spasmodic movements of the face due to habit spasm or spasmodic tic. In these latter the movements are often partly bi-lateral, and of the nature of grimaces, and may be part of a more generalised spasmodic tic affecting the trunk and limbs. There is in addition a bi-lateral *blepharospasm* or spasm of the orbicularis palpebrarum on both sides, which may originate from emotional disturbances, and is then unaccompanied by any clonic spasm of the remaining facial musculature, or of the limbs.

I have, for instance, seen such bi-lateral clonic blepharospasm originate in a lady of 55 after long worrying over her favourite daughter's marriage and departure for India. In such cases it is a psycho-motor reflex, of the nature of crying, though lachrymation does not accompany the spasms.

Clonic spasm of part of the facial musculature is also seen occasionally as a sequel of encephalitis lethargica. Spasms, moreover, of the whole side of the face may recur at intervals from pressure on the nerve by a cerebellar tumour. Twittering contractions in the orbicularis palpebrarum on one side are of common occurrence in numerous individuals, and are apt to be brought on by fatigue, especially of mental origin.

These spasms are not sufficiently powerful to close the eye completely, but may be sufficiently strong to interfere slightly with vision, the individual spasms persisting for several seconds. These may occur scores of times during the day, sometimes for weeks at a time, but do not necessarily lead to the development of the fully developed type of clonic facial spasm. They are known by the terms "live blood," or "bird in the eye," and may be temporarily arrested by

rubbing the eye or by the application of moderately strong faradism over the closed eye.

True Clonic Facial Spasm is a disease of adult life commencing usually after 40, though I have known it to commence in a girl of 20, after extraction of an upper wisdom tooth, the spasms originating in the orbicularis palpebrarum, and gradually spreading to all the facial muscles on that side. At its origin the movement is usually limited to the orbicularis palpebrarum and is not then to be distinguished from the slighter form lately referred to as "live blood." Instead of disappearing, after a time the intermittent spasms begin to affect other muscles as well, so that the zygomatici and risorius pull on the naso-labial fold and angle of the mouth, while the eye may be almost completely closed by the orbicularis spasms. After the lapse of perhaps a year or two, more facial muscles on that side are attacked by the spasms until every muscle supplied by the facial nerve will be thrown into contraction, just as if the electrode of a faradic battery had been placed over the facial nerve beneath the mastoid. Such strong spasms as these interfere very much with the patient's comfort and are apt to attract the attention of bystanders, and to obscure vision by the closure of that eye. Occasionally spasm of the orbicularis palpebrarum induces reflex spasm of the other eye also, so that with strong closure of both eyes vision is seriously interfered with, and it may be even dangerous to the patient, as when walking in traffic.

When the clonic spasms are strong on one side and have lasted a number of years, slight clonic spasms may be seen in the facial musculature on the opposite side also, though I have never seen this sufficiently serious to warrant bilateral treatment, except very occasionally of the two orbiculares palpebrarum. After the facial spasm has persisted for several months or years it will invariably be found that slight weakness of the facial movements is present on strong voluntary effort, indicating slight progressive damage of the motor fibres.

The cause of these clonic facial spasms is unknown, though it has been suggested that it is an irritative lesion of the geniculate ganglion. I have seen a number of cases of clonic facial spasm accompanying chronic trigeminal neuralgia of the same side, and this simultaneous motor and sensory tic is suggestive of some common factor. Indeed Cushing has described a rare form of severe facial neuralgia combined with severe clonic facial spasm which is not relieved by Gasserian operation, though one at least of his cases was relieved by section of the facial nerve, a

Fig. 44.—Idiopathic clonic facial spasm, right side. The spasms were completely arrested by alcohol injection of the nerve at the stylo-mastoid foramen, causing partial facial paresis.

point which is suggestive of the pain being associated with the seventh or facial nerve system rather than the trigeminal, and it might therefore be classed as a form of geniculate neuralgia. In the cases which I have seen, however, of true clonic facial spasm associated with trigeminal neuralgia of the same side, some half a dozen in number, the facial spasms and the neuralgia appeared to be mostly independent of each other, in that alcohol injection of the deep foramina cured the trigeminal neuralgia, but did not influence the clonic spasms, which, with one exception, persisted. These spasms must, of course, be differentiated from the reflex grimacing and screwing up of the face which is liable to accompany a severe spasm of neuralgia.

The treatment of clonic facial spasm is practically limited to weakening of the facial nerve by exposure and stretching of the nerve, or, better still, by alcohol puncture at the stylo-mastoid foramen. To reach this foramen, which is the exit of the facial nerve from the skull and lies at the root of the mastoid process, a fine needle one and a half inches in length must be sunk behind the pinna just in front of the tip of the mastoid process. The best line for the direction of the needle is obtained by drawing two vertical lines with a skin writing pencil behind the ear, one at the tip of the mastoid and one below the posterior border of the external auditory meatus; a point midway between these two lines is taken at the level of the tip of the mastoid, and the needle is pushed upwards at an angle of about 45° in the plane perpendicular to the side of the head drawn through this point. The foramen will be found at a depth of from one to one and a quarter inches, when a preliminary injection of two or three minims of novocain solution is made. If within half a minute definite facial paresis is noticeable the syringe is changed carefully, while the needle is held stationary, and three or four drops of 90 per cent. alcohol are injected. At once practically complete facial paresis will be seen. If no more than three or four minims of alcohol have been injected the facial paresis will diminish in severity after the lapse of a few days, and the face may be nearly straight when at rest, though the clonic facial spasms have entirely disappeared and may not recur for six months.

If the upper branch of the facial nerve is more affected by the clonic facial spasms this portion may be picked out by puncturing with a hypodermic needle at the back of the parotid region at the level of the tragus, while the orbicularis palpebrarum alone may be treated by hypodermic alcohol puncture along the outer rim of the orbit at the level of the external canthus. These alcohol punctures are likely to require renewing every six to twelve months, but the relief from spasm which they give is valuable.

CHAPTER XIX

OTHER CRANIAL NERVES (VIII—XII)

Eighth or Auditory Nerve.—The auditory nerve arises from two nuclei in the side of the medulla, the ventral cochlear nucleus receiving the nerve of hearing from the cochlea, continuing as the striæ acusticæ, which are possibly the endings of the nerve of hearing proper, which run transversely across the floor of the fourth ventricle. The vestibular nucleus receives the fibres of the vestibular portion of the auditory nerve, which enter the medulla on the ventral side of the restiform body. The auditory nerve thus consists really of two nerves, the cochlear nerve and the vestibular nerve, the former ending in the organ of hearing in the cochlea, while the vestibular nerve is distributed to the labyrinth. The fibres of the vestibular nerve after reaching the vestibular dorsal nucleus are connected with the cerebellum and with the superior olive and sixth nerve nuclei. Impressions received from the labyrinth have thus direct influence upon the co-ordinating centres in the cerebellum, and on the conjugate movement of the eyes. Hence it is that in early labyrinthine vertigo, or the syndrome of Ménière's disease, sudden afferent stimuli starting from the labyrinth produce intense vertigo and inco-ordination, so that the patient may fall, and at the same time objects may appear to move, owing to the sudden appearance of nystagmus. This condition is a common sequel of chronic sclerosis of the middle ear, which spreads into the labyrinth and inner ear, causing deafness.

Indeed the mere pressure of wax upon the tympanum may produce much the same symptoms. Tinnitus is a usual accompaniment of chronic labyrinthine sclerosis, a constant buzzing in the ear or a noise like the singing of a

kettle, or sometimes the shrill note of a train whistle being heard. The tinnitus is more or less constant, but the vertigo is usually paroxysmal, the tinnitus as a rule being louder with the onset of the giddiness. Vomiting may accompany the attack, and even loss of consciousness has been described, though this is rare.

The auditory nerve may be damaged by fracture of the base of the skull, or it may be pressed upon by cerebellar or other tumours of the base. In a woman suffering from a tumour growing upon the fifth nerve which pressed upwards on the cerebellum and backwards on the auditory nerve there was marked tinnitus produced, the sound being described as of the roaring of a cataract, so that when her eyes were closed she could almost believe she was back in her old home in Canada, not far distant from the Falls of Niagara. Tumours may grow on the sheath of the auditory nerve itself close to the internal auditory meatus, usually of the nature of fibroma or fibro-sarcoma. These tumours may be uni-lateral or bi-lateral, and they may be associated with neurofibromata on the posterior spinal roots, or even with multiple subcutaneous neurofibromata or von Recklinghausen's disease. These fibromata of the auditory nerve sheath gradually destroy the auditory nerve by pressure, causing nerve deafness, usually preceded by tinnitus. These tumours being situated in the ponto-cerebellar angle cause upward pressure on the cerebellum, and consequently symptoms of cerebellar disease will be a feature in these cases. Headache may not be present until the symptoms have lasted many months, though ultimately œdema will be produced and the symptoms of increasing intra-cranial pressure will appear, namely, headache, vomiting and optic neuritis or papillœdema. These cases may be diagnosed in the earlier stages by the combination of the symptoms of ataxic gait, inco-ordination of one or even both arms, with increasing nerve deafness and tinnitus, giddiness and nystagmus. These auditory fibromata differ from other neuromata in microscopical section, the fibres showing a palisade arrangement.

Neuritis of the auditory nerve may be due to toxic causes, either of infective origin or due to certain chemical poisons, such as lead, arsenic and quinine. Temporary deafness is a well-known symptom from an overdose of quinine, often accompanied by tinnitus. Salicylates in larger doses also produce the same effect. The non-advisability of employing quinine even in small doses, unless absolutely necessary, in cases of chronic deafness is well known. Influenza may be followed by deafness, possibly a symmetrical auditory neuritis, and syphilis in the secondary stage occasionally produces symmetrical auditory neuritis with total deafness, sometimes accompanied by bi-lateral facial paralysis. In the treatment of syphilis overdosage or too frequent repetition of arseno-benzol injections may give rise to tinnitus or other symptoms of arsenical poisoning.

The auditory nerves, too, may be involved with many other cranial nerves in the condition known as polyneuritis cranialis, but this is rarely, if ever, a symmetrical toxic multiple neuritis, the cranial polyneuritis being usually due to an inflammatory mass at the base of the brain secondary to chronic middle ear disease, or to caries of the skull, accompanied by chronic meningitis, or to basal tumours.

Glosso-pharyngeal and Vagus Nerves.—These two nerves are perhaps best considered together. They consist of both sensory and motor fibres, the sensory fibres of the glosso-pharyngeal taking origin from a ganglion of nerve cells known as the petrous ganglion, while those of the vagus arise from a similar jugular ganglion. Entering the medulla on the dorsal side of the olive a portion of the glosso-pharyngeal fibres pass into the solitary bundle which runs upwards surrounded with nerve cells to a point not far from the sensory nucleus of the trigeminal. Into this collection of nerve cells in which the nerve fibres of the solitary bundle end, there terminate also fibres from the trigeminal nerve and the pars intermedia Wrisbergi, and this nucleus of the solitary bundle is sometimes known as the gustatory nucleus.

Other glosso-pharyngeal fibres with sensory vagus fibres terminate in nuclei considerably lower. The motor fibres of these two nerves arise from a nucleus called the nucleus ambiguus, which lies ventral to the vestibular nucleus and dorsal to the olive. These motor fibres supply the constrictors of the pharynx and the soft palate, while the intrinsic laryngeal muscles are supplied by means of the recurrent laryngeal, which leaves the vagus nerve within the chest, winding round the aorta on the left side and the subclavian artery on the right. The *superior laryngeal* nerve, or sensory nerve of the larynx, is given off from the vagus below its exit from the skull at the jugular foramen, passes downwards by the side of the pharynx behind the internal carotid artery, and divides into external and internal laryngeal branches. The former supplies the crico-thyroid muscles, while the internal branch passes through the thyro-hyoid membrane to supply the mucous membrane to the larynx. At the point where the internal laryngeal branch pierces the thyro-hyoid membrane the nerve may sometimes be successfully injected with alcohol in cases of intractable pain due to chronic tuberculosis of the larynx. Paralysis of the external branch which supplies the crico-thyroid muscle causes hoarseness of voice.

The *recurrent laryngeal* nerves which supply the intrinsic musculature of the larynx, with the abductor and adductor muscles of the vocal cords, are liable to damage within the thorax, the nerve on the left side being subject to compression by aneurysm of the aorta, or other mediastinal tumours. I have seen it paralysed from pressure of a dilated left auricle in mitral stenosis in a man I showed at a meeting of the Harveian Society in 1902, and a few other similar cases have been published. As a result the left vocal cord will be paralysed, being semi-abducted in the so-called cadaveric position. In the stage of partial paralysis the vocal cord will lie in the middle line, the abductor muscle always being paralysed before the adductor. In the early stage the voice may be scarcely altered, as the cords will meet in the middle line during phonation, but when one

cord is completely paralysed the voice becomes hoarse or almost whispering and an explosive cough is no longer possible. In aortic aneurysm the cervical sympathetic nerve is liable to be paralysed at the same time as the left recurrent laryngeal, owing to its fibres forming a ring around the aorta, known as the annulus of Vieussens. Sympathetic paralysis produces slight ptosis, and a medium small pupil which reacts to light normally, except that it dilates only slightly when shaded. On the right side the recurrent laryngeal winds round the sub-clavian artery, and is not liable to damage from tumours within the chest. The two recurrent laryngeal nerves run upwards on each side between the trachea and œsophagus, and here may be damaged by stab wounds or by the pressure of enlarged glands.

Peripheral paralysis of the recurrent laryngeal nerve within the larynx may occasionally occur in polyneuritis, especially as the result of chronic lead poisoning. The condition produced will be bi-lateral abductor paralysis, and the voice may be scarcely altered, though inspiratory stridor is likely to be present, especially during sleep, and acute dyspnoea may be produced by slight congestion of the cords from laryngitis, or the œdema of the glottis that occurs in chronic Bright's disease.

Such urgent dyspnœa may require tracheotomy. Tumours at the base of the skull or enlarged glands at the jugular fossa may damage the vago-glosso-pharyngeal, producing uni-lateral paralysis of the palate and vocal cord, while lesions of the recurrent laryngeal will affect the vocal cord only without the palate. If therefore palatal palsy accompanies vocal cord paralysis, it is useless to look for the lesion within the thorax, but the source of the mischief must be looked for at the base of the skull, or in the nucleus ambiguus. Vago-glosso-pharyngeal motor paralysis through causing paralysis of the palate and constrictors of the pharynx will produce difficulty in swallowing, and nasal regurgitation. Uni-lateral paralysis of the palate will cause slightly nasal voice, with occasional regurgitation of liquids, while on

inspiration the palate will be seen to hang lower on the affected side, and on phonation the raphé will be drawn over to the sound side, the arch of the soft palate being raised distinctly higher than on the affected side. Certain words and consonants will bring out the nasal speech better than others, for example, rubber, head and egg become "rummer," "hen," and "eng." When both sides of the palate are paralysed the velum hangs down motionless, and if the cheeks are blown out with the lips held tight and the nostrils compressed, sudden release of the nostrils causes a blast of air to appear through them, owing to the inability of the soft palate to shut off the posterior nares.

Before giving off the recurrent laryngeal nerves the vagus gives branches in the pharynx to the cardiac plexus, and piercing the diaphragm reaches the solar plexus and supplies motor fibres to the stomach and to the upper half of the intestines. Vagus paralysis will cause cardiac arrhythmia and tachycardia, but bi-lateral vagus paralysis, produced experimentally in animals, causes rapid onset of pneumonia and death. A well-known sensory branch of the vagus, the auricular branch or *Arnold's nerve*, arises from the jugular ganglion of the root, and after communicating with the facial nerve it reaches the surface to supply the skin of the ear at the back of the pinna. Owing to a habit of gastronomic experts, towards the end of a City dinner, of rubbing the skin behind the ear with a moistened napkin with a view to stimulating the digestive process, Arnold's nerve has been named the "Alderman's" nerve.

The sensory supply of the glosso-pharyngeal is distributed to the upper part of the pharynx, the posterior portion of the tongue behind the circumvallate papillæ, the posterior border of the soft palate and uvula, the tonsil and epiglottis and eustachian tube, whilst its tympanic branch, or *nerve of Jacobson*, arises from the petrous ganglion, and enters the tympanum, communicating with the great superficial petrosal nerve, and Meckel's ganglion, and also by one of its branches, the small superficial petrosal, with both the geniculate and otic ganglia. With the deep petrosal

branches from the sympathetic this plexus is known as the tympanic plexus, forming a communication between the second and third divisions of the fifth nerve, the seventh, glosso-pharyngeal and sympathetic nerves. Paralysis of the glosso-pharyngeal from neuritis or by pressure of growths or caries of the skull will produce anæsthesia of the pharynx, loss of the pharyngeal reflex, and loss of taste at the back of the tongue. The glosso-pharyngeal, like the vagus and indeed other cranial nerves, is frequently affected by neuritis subsequent to faucial diphtheria (see p. 21).

Neuralgia of the glosso-pharyngeal may be produced by growths in the tonsil and pharyngeal wall. I have seen severe paroxysms of violent pain produced by light touches on the upper part of the neck and behind the angle of the jaw in cases of recurrence of growth secondary to laryngeal cancer and cancer of the tongue. Idiopathic neuralgia of the glosso-pharyngeal or true spasmodic tic douloureux closely resembling chronic trigeminal neuralgia in character is a rare disease, of which I have seen but three cases (see p. 327).

Spinal Accessory Paralysis. — The accessory portion has already been described under vago-glosso-pharyngeal nerve. The spinal portion arises from the ventro-lateral portion of the anterior horns, chiefly of the three upper cervical segments, though their nuclei can be traced as far as the sixth cervical. These fibres run upwards into the cranium to join the accessory nerve at the jugular foramen, and are distributed ultimately to the sterno-mastoid and trapezius muscles. Upper cervical lesions such as gummatous meningitis or tumours, poliomyelitis, caries of the base of the skull, or growths in the region of the jugular foramen will be liable to paralyse the sterno-mastoid and trapezius muscles, though it is only from lesions at the jugular foramen that the palate and vocal cord, together with the sterno-mastoid and trapezius, will all be paralysed at the same time.

The spinal-accessory nerve is liable to damage in the

upper part of the neck behind the angle of the jaw from suppurating glands or from pressure of a tumour, or from an operation for tuberculous glands. The nerve may also be injured in this region by nerve anastomosis for facial paralysis, and as a result there will be paralysis of the sterno-mastoid and trapezius muscles. The nerve enters the sterno-mastoid muscle near its anterior border about a quarter of the length of the muscle below its origin from the mastoid process. The nerve traverses the muscle in an oblique direction downwards and backwards, giving branches to supply the muscle, and leaves the muscle at its posterior border about its middle. Traversing the posterior triangle the nerve then enters the trapezius muscle, which it supplies. Stab wounds in the neck may damage the nerve, or it may be wounded or cut in an operation for the removal of tuberculous glands or malignant growth in the posterior triangle (see p. 68).

Hypo-glossal Nerve.—This nerve arises from a well-defined nucleus of large cells close to the raphé in the medulla, and its fibres emerge on the ventral side of the olive, between that structure and the pyramid, differing from the other cranial nerves which emerge on the dorsal side of the olive. This nerve, therefore, closely resembles an anterior spinal root both in its anatomy and function. Within the medulla the hypo-glossal nucleus may suffer from tumours such as glioma or tuberculoma, while nuclear paralysis may be acute, as in poliomyelitis, or chronic, as in bulbar paralysis accompanying amyotrophic lateral sclerosis. Syringomyelia too may not uncommonly damage the hypo-glossal nucleus, often on one side only, producing hemiatrophy of the tongue. After emergence from the medulla the hypo-glossal roots may suffer from gummatous meningitis or malignant growths, from pressure by aneurysm of the vertebral artery, or by tuberculous caries of the base of the skull. Leaving the skull by the anterior condyloid foramen the nerve may suffer from fracture or fracture dislocation of the atlas vertebra, and when the nerve reaches the angle of the jaw it is liable, like the accessory

nerve, to be damaged by tumours or suppurating glands, or to suffer by the operation of facio-hypo-glossal anastomosis for the treatment of facial paralysis. Injury to the hypo-glossal nerve on one side will cause hemiatrophy of the tongue, which becomes narrowed, shrunken and wrinkled, and cannot properly be protruded, the end curling towards the wasted side. Fibrillary tremors of the muscle are seen in chronic nuclear degeneration in bulbar paralysis.

Recovery has been seen after hemiatrophy of the tongue when due to pressure of tuberculous glands, or gummatous meningitis, but in bulbar palsy and in syringomyelia the atrophy, once developed, is permanent.

CHAPTER XX

VISCERAL NEURALGIAS

Although in normal conditions we are scarcely aware of afferent impressions received from the various viscera such as the heart, lungs, liver, kidneys, etc., yet it is an undoubted fact that in various disorders of these organs these afferent impressions may be so much intensified as to rise above the threshold of consciousness so that we are made aware of sensations varying in intensity from a dull ache to the most intolerably severe pain. As examples of slight discomfort may be quoted the sensation of gastric distension from flatulence or after a full meal, and the sensation of heavy weight and aching round the right ribs and shoulder blade in disorders of the liver. Shoulder-tip pain is a well-known reflex neuralgia through the phrenic nerve and the cervical plexus, produced by diaphragmatic inflammations, sub-phrenic abscess and gastric perforation. A chronic appendix and duodenal or gastric ulcer may cause slight aching and local tenderness, while at other times the pain from these causes may be most intense. Other examples of severe visceral pain associated with diseases of the organs concerned are angina pectoris, renal and biliary colic, and pleurisy. In angina pectoris there is no doubt muscular cramp of the heart muscle, and of the circular muscles in the bile duct or ureter in the two forms of colic.

The source of the pain in these cases of gross disease of the viscera is comparatively obvious, and the term neuralgia as applied to these pains is best avoided, and should be reserved for those instances of pain referred to an organ in which organic disease cannot be demonstrated. Thus, neuralgia of the liver or of the kidneys has been described

as persisting for years when there was no evidence of disease of either of these organs.

Renal crises of violent pain resembling renal colic, and known as Dietl's crises, may recur at intervals in cases where no stone or other organic lesion can be demonstrated, though they are apt to be associated with visceroptosis, where the kidney is exceptionally movable. It has been suggested that some kinking of the pedicle of the kidney may account for the attacks. In such cases the use of an abdominal belt as a support for the kidney is advisable.

Acute crises of abdominal pain associated with vomiting are occasionally a feature of migraine, the abdominal pain being really a substituted neuralgia; such cases may be called cœliac neuralgia, though it is doubtful whether the pain is limited to this nerve area. In locomotor ataxy recurring paroxysmal attacks of gastralgia and vomiting may cause the most violent pain and acute distress.

No local lesion of the stomach is known to be the cause of these attacks, though in three fatal cases I found the most enormous gastric dilatation, the stomach appearing to fill the whole abdomen.

True referred neuralgias associated with visceral disease were described by Hilton in 1863 and in much greater detail by Head in 1892, in which local areas of tenderness may be found over a posterior root cutaneous distribution, which is supplied by the same segment of the spinal cord as supplies the particular viscus involved by some gross disease. Thus, gastric ulcer usually affects the pyloric end of the stomach, and consequently referred neuralgias and hyperæsthesia of the skin associated with such ulcers will be found over the skin areas supplied from the lowest spinal segment which receives sensory nerve fibres from the pyloric end of the stomach. It is especially the 7th, 8th, and 9th dorsal spinal segments which supply the stomach with afferent fibres, and sometimes no doubt also the sixth dorsal supplies the cardiac end; pyloric ulcer will therefore be associated with cutaneous hyperæsthesia and tenderness of the skin of the abdomen

chiefly on the left side between the xiphisternum and the navel, and sometimes when the ulcer is on the posterior wall of the lesser curvature the patient will complain of pain more especially in the lower dorsal region of the back, perhaps radiating through to the front. In a case of gastric pain associated with dense fibrous adhesions between the cardiac end of the stomach and the spleen I have seen excessive cutaneous tenderness with referred pain below the left breast. The testes and ovary also may be the seat of troublesome chronic neuralgia, and it is more particularly pain associated with these organs which may take on the appearance of a psychalgia or mental pain. *Testicular neuralgia* is certainly more apt to occur in neuropathic individuals; the pain is usually of a dull dragging nature, but may sometimes be acute and associated with cramps in the legs and vomiting.

In severe cases this may be treated by section of the testicular nerve, though this operation is best avoided as long as possible, and the use of a suspensory bandage should be encouraged.

Left-sided varicocele is also a common source of discomfort and even pain in nervous men, the discomfort being often quite out of proportion to the size of the varicocele. For this also a suspensory bandage should be worn and the patient should be encouraged to distract his mind as far as possible from this region, as neuralgias and other abnormal sensations referred to the generative organs are especially apt to cause mental depression and troublesome obsessions (see p. 135).

Ovarian Neuralgia is usually associated with hyperæsthesia of the skin just above the groin on one side, with intense tenderness of the ovary on palpation. This condition is usually associated with neurasthenic symptoms, and is apt to be aggravated just before the menstrual flow. I have also known abdominal pain below the navel to recur daily for some days, for half an hour to two hours at a time, until menstruation started. Of two sisters, one *æt.* 13, who had not yet commenced menstruation, had for

four months been suffering almost daily for an hour or two from attacks of abdominal pain referred across the lower abdomen. During that period she was free from pain for two or three weeks only, while between the attacks she was quite well, and there were no signs to suggest appendix or other mischief. Her elder sister had suffered in exactly the same way for two years, the pain ceasing at the age of 16, when the catamenia started. These pains undoubtedly may be looked upon as an *ante-menstrual neuralgia*, though whether connected more directly with the uterus, tubes or ovaries I am uncertain, and the possibility of such a neuralgia should be borne in mind in young girls complaining of abdominal pains, before deciding upon operating upon a possibly blameless appendix. The mother of these two girls was also a neuralgic subject and had suffered from paroxysmal supra-orbital neuralgia on both sides at different times.

Another girl aged 12½ had for some months suffered from recurring pain in the lower abdomen and left side with frequent nausea, and slight tenderness in the middle line below the navel. The pain improved under bromide and extract. cimicifug. liq. and later disappeared entirely when the catamenia became established.

A woman aged 24, married sixteen months, with no child, and of nervous temperament, was much upset at the death of her father and began to suffer from pains of sudden onset in the lower back and abdomen, accompanied by nausea but no vomiting. She described her pains as starting like a needle below the navel, spreading between the thighs and down the inside of the groin. She was of a restless temperament, talking much in her sleep and given to dreaming a good deal. The diagnosis of uterine neuralgia appeared to be confirmed by the disappearance of the pain after dilatation of the cervix had been done.

Spinal Neuralgias due to Bone Disease

Chronic pain in the back of the neck and often in the limbs and round the ribs may be due to various forms of disease

affecting the vertebral column. Osteo-arthritis, already referred to as being a cause of occipital neuralgia, may in the same way affect any spinal nerve and set up radiating pains along its course. The diagnosis of this condition will be suggested by rigidity of the spine and pain on movement, by other evidences of osteo-arthritis or chronic rheumatism, and lastly by X-ray photographs.

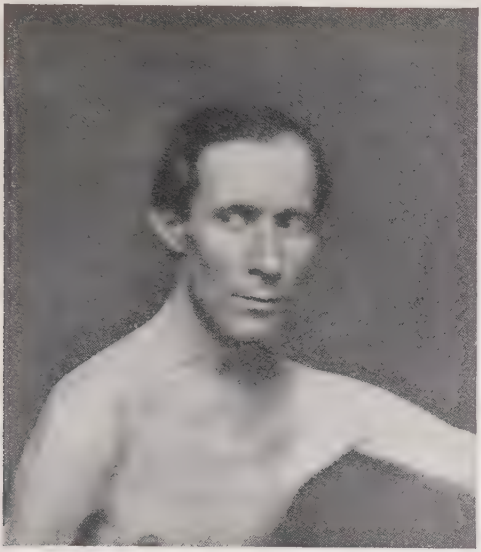

FIG. 45.—Case of Spondylose Rhizomélique. The head is turned to the right as far as possible, and the left arm raised, to illustrate the fixity of the cervical spine and shoulder joint. The spine is also fixed in an antero-posterior curvature.

These latter should always be taken stereoscopically, and the actual negatives examined in a suitable stereoscope, when the view obtained of a solid transparency of the spinal column is infinitely more valuable in localisation and recognition of disease than is a simple flat negative.

Other forms of spondylitis such as osteitis deformans, or Paget's disease of the bones, and spondylose rhizomélique, a form of chronic spondylitis with rigidity of the whole spine and affection of some of the proximal joints, such as the

shoulder and hip joints and sterno-clavicular joints, are likely to be the source of chronic pains in the back and neck.

In the latter disease the whole spine is curved antero-posteriorly and is quite rigid, while the head can scarcely be moved sideways, and the movements of the limbs at the hips and shoulders are restricted. The clinical picture of this disease is thus easily recognised and the pains associated with it explained.

Much more puzzling sometimes may be the pain in the back in the early stage of Pott's disease or tuberculosis of the vertebræ, malignant disease, aneurysm, hydatid and other tumours.

I have known tuberculosis of the spine in the middle dorsal region cause such severe pain in a young soldier, long before the War, that he received severe punishment for having fallen out on parade because of the pain; malingering was the diagnosis made until weakness of the legs commenced and a well-marked curvature of his back was found. In his case intense spastic paraplegia developed; this was ultimately relieved by laminectomy and the evacuation of a tuberculous abscess pressing upon the spinal cord. In other cases of Paget's disease or in tumour of the spine the pain may not be referred locally in the back but may be referred to the front of the chest or pit of the stomach. Girdle pain may not be complained of and the pain in front is liable to be mistaken for visceral disease in the chest or abdomen.

Aortic aneurysm too may erode the upper dorsal vertebræ and cause either pain in the back or girdle pain. Hydatid of the spine is very rare in this country, though in the Antipodes and cattle-raising countries it is as common as tuberculosis.

Many years ago I saw a case of hydatid disease of the lumbar vertebræ in a man of about 45 whose complaint for seven months was of severe pain in the back. This was mistaken by a very famous physician for hysteria, as no physical signs could be found to explain the pain.

This was in 1895, before X-rays were known, and he was

treated as a functional case until one day he developed an acute transverse myelitis at the level of the fourth lumbar segment. Laminectomy then evacuated the hydatid cyst, but was too late to improve his condition of permanent paralysis from the myelitis.

One must be strictly on one's guard against what may be called a natural tendency to diagnose pains as "functional" for which no adequate real reason can be found. Such a diagnosis is all the more likely to be made when the patient shows signs of a nervous temperament. Yet it must constantly be remembered that persistent suffering engenders the development of neurasthenic symptoms, and anyone who has to suffer frequent or persistent pain is liable to become irritable, jumpy and despondent, all the more so if the doctor in attendance, and through him the patient's friends, disbelieve the reality of his pain and look upon it as hysterical. More cruelties perhaps are perpetrated by faulty diagnosis and the non-recognition of spinal neuralgia when due to organic disease than in any other form of illness.

Secondary malignant growths, such as are often the sequel of breast cancer or uterine malignant growth, are very frequent sources of persistent spinal pain, and chronic pain in the back or legs, following within a few months or even two or three years after an operation for malignant disease, should be looked upon with grave suspicion. Very frequently the disease attacks the upper lumbar vertebræ and the pains resulting will follow the sciatic distribution. Bi-lateral sciatica should always be closely suspected as being probably due either to central spinal disease or to some cause of toxæmic neuritis, such as diabetes. The spinal column should always be carefully examined for any evidence of angular curvature or undue prominence of a vertebral spine. Local tenderness on pressure over the vertebræ must also be sought for, and when this is present in association with diminished mobility of the spinal column the diagnosis of bone disease is probable. The spine must be examined for its mobility both antero-posteriorly and for lateral movement, and if

its movement is free and unimpaired actual bone disease may be practically excluded.

Local tenderness on pressure or percussion of the spines is often a very prominent symptom in neurasthenia, known as neurasthenic spine, but in this case mobility of the spinal column is always normal.

Rigidity should always make one suspicious of some form of spondylitis or new growth. It is true that in late middle life a general antero-posterior curvature may be found in men whose occupation necessitates much stooping, such as gardeners or carriers of heavy packs, and the pains of chronic rheumatism may sometimes be difficult at first to distinguish from local disease of the spinal column due to caries or carcinoma.

Sciatica in Malignant Disease of the Spine

Pains radiating into the legs from the hips and back, intermittent and varying in severity, and occurring within a year or two after an operation for carcinoma, almost invariably herald the development of secondary growth, either in the bones of the lumbo-sacral region or else of secondary deposits in the pelvis in the neighbourhood of the intervertebral foramina, whence the anterior branches of the spinal nerves emerge on their way to the lumbo-sacral plexus. Such sciatic pains may spread down the limb to the ankle and foot, as in ordinary sciatica, and after a time may be followed by muscular wasting in the leg and numbness of the foot. The Achilles jerk will then probably be absent, though the knee jerk may be present, and extension of the nerve by flexion of the hip with the knee kept straight will not specially aggravate the pain, as is commonly the case in ordinary sciatic peri-neuritis. In some cases, the pains do not spread far below the knee, though perhaps they reach the inner side of the ankle without extending into the foot; the pain is then more usually round the groin and front of the thigh than down the back of the limb.

In such cases, it is the anterior crural nerve which is more particularly involved, and the knee jerk will probably be

lost, and some wasting of the quadriceps will be noticeable. Bi-lateral sciatica is always a suspicious sign of gross central disease of the spine or the spinal cord. A curious feature of the sciatic pains met with in secondary malignant disease which I have often noticed is that the pain may attack one limb first severely, and may then disappear from that side to attack the opposite limb. With a history of previous operation for malignant growth, such as of the breast or uterus, the appearance of such sciatic pains unrelieved by heat, or other remedies that may be useful in the rheumatic type of sciatica, will make the diagnosis of secondary disease almost certain.

X-rays may confirm the diagnosis, but evidence of bone involvement will not always be visible. Such patients are always much more ill in appearance, worn-looking and perhaps anæmic, more apt to keep to their bed and avoid the pain produced in the back by jarring from the foot touching the ground, than are sufferers from sciatic perineuritis; there will be usually little difficulty in deciding between the two conditions, even though, as sometimes occurs, rheumatic sciatica may be bi-lateral.

Treatment of what may be called " malignant sciatica " is entirely palliative. Rest in bed or on a couch and the avoidance of walking or standing is practically essential. Analgesic drugs, such as aspirin and pyramidon, in combination with medinal and heroin at night, will help to keep the pain at bay, or codeine or omnopon may be used instead of heroin.

Usually local radiant heat or ionisation aggravate the pain. Failing relief from other remedies, injections of morphine may ultimately have to be resorted to, and no hesitation should be permitted in employing this remedy for the relief of the severe pains of this disease. I have known persons delay or even refuse to give morphine injections to such patients because of the risk of their acquiring the morphine habit! A little reflection will show that such a habit cannot be of any serious harm in this condition, and life may undoubtedly be prolonged through its use.

CHAPTER XXI

CHRONIC DISEASE OF THE SPINAL CORD

In locomotor ataxy, or tabes dorsalis, one of the most constant symptoms, and the initial symptom in over 60 per cent. of cases, is the frequent recurrence of shooting or lightning pains. These neuralgic pains usually involve the legs, but occasionally may be felt in the trunk and even in the arms. They are frequently spoken of by the sufferers themselves as "neuritis," and a term that has often been applied to them is *tabetic neuralgia*. The most characteristic feature of the pain is its sudden sharp appearance and fleeting duration, appearing to flicker up and down the limb like electricity or lightning. These pains often vary with the weather, and are apt to be worse with a low barometer before the appearance of rain and with cold east winds. They may be present over a large area, and vary much in severity. Often the neuralgic crises are sufficiently intense to make the patients scream out with agony, while at other times they may be comparatively free for weeks or even months. Any debilitating febrile illness like influenza is likely to aggravate the pains.

The diagnosis of the condition as tabes will firstly be suggested by the shooting character of the neuralgic pains, and when in addition analgesia, or loss to pin prick, is demonstrable on the legs, with perhaps loss of knee jerks or Achilles jerks on one or both sides, and Argyll Robertson pupils, or loss of the pupil reaction to light, the diagnosis may be considered established. Although there may be loss to pin prick on the legs and lowered sensibility to heat, yet tactile sensation may be fairly good and the sense of vibration unimpaired. Deep sensibility

to hard squeeze of the calf muscle will evoke no pain, as it would in a normal subject. When standing with the feet together and eyes closed—known as Romberg's test—there may be no unsteadiness. Hyperexcitability to cold on the legs and trunk is not uncommon, though it is surprising to find excessive shrinking on contact with cold metal over areas where sensibility to pin prick and to heat is considerably diminished. The term psychroæsthesia has been used to express this excessive sensitiveness to cold.

The treatment of tabetic pains will be both symptomatic and also curative. Being presumably due to chronic syphilitic root neuritis affecting the posterior roots between the root ganglion and the spinal cord, anti-syphilitic remedies should always be tried, and mercury be given either by the mouth as biniodide or by injections intramuscularly as grey oil or calomel cream, or else by intravenous injections as small doses of cyanide of mercury. Some practitioners still advocate inunctions of mercury to the skin daily for thirty-six or more rubbings, but it is a messy treatment and leaves uncertainty as to the amount absorbed, and it is being largely replaced by more modern methods of treatment. In addition to the employment of mercury and iodide, the intra-venous employment of arseno-benzol preparations is of great value. Thus two or three intra-venous injections of 0·6 gramme of salvarsan, or its British equivalent kharsivan, at intervals of ten days to a fortnight, sometimes have a most remarkable effect in reducing lightning pains, though occasionally the pains may be made much worse for the first twelve hours or more. In addition to the intra-venous injections of salvarsan intra-thecal injections of auto-serum may be made, and I have seen repeatedly the greatest benefit to the pains result from the administration of intra-thecal injections of auto-serum, following salvarsan given intravenously. The method was introduced first by Swift and Ellis of New York, who called it salvarsanised serum, and the method I employ is modified from theirs. First,

an intra-venous injection of 0·6 gramme salvarsan is given, and fourteen days later this dose is repeated, and immediately afterwards about 5 ounces of blood are withdrawn from the vein into a sterilised flask which is carefully sealed and put away in a cupboard at room temperature. Some eighteen hours afterwards the blood will be found to have clotted well, and the clot has contracted, giving an exudation of straw-coloured serum; this is carefully decanted and warmed to blood temperature by standing in hot water. Lumbar puncture is then done, and if the cerebro-spinal fluid flows readily it is allowed to do so until the pressure falls, when the serum is run into the intra-thecal space through a sterilised burette and tube to the amount of 50 to 55 cubic centimetres. No special precaution need be observed of allowing an equal quantity of cerebro-spinal fluid to escape before introducing the serum. If the fluid flows readily I allow it to escape, but if it flows only drop by drop it is then unnecessary to wait, and the serum should be run in at once. The first 30 cubic centimetres may be run in quite quickly without any symptoms of head pressure being produced, but the last 20 cubic centimetres must be run in much more slowly, and if any head pressure is complained of by the patient a little fluid should be allowed to flow back into the funnel, and no more should be introduced for two or three minutes.

After completing the injection the patient must be kept in bed for two days. In the majority of cases shooting pains will commence in the limbs within one and a half to two hours after the completion of the injection, and the severity of the pains is usually greater than at any previous time. This attack may last twelve hours or even longer and may require repeated injections of morphine to bring relief. The pains then disappear completely and may not return except very slightly for two years or more, and even then with much-reduced severity. The large majority of severe tabetic shooting pains that I have seen treated by this method have benefited greatly, and not only the tabetic neuralgia, but also other symptoms of

tabes may improve at the same time. It is only in cases of tabes that the intra-thecal injections of serum provoke any pains in the limbs, and it is no doubt due to a congestive process set up in the meningeal coverings of the damaged posterior roots that causes the severe pains for several hours with the subsequent improvement.

Disseminated Sclerosis

Disseminated or insular sclerosis is a chronic cerebro-spinal disease of uncertain origin. Many cases undoubtedly follow closely upon injury such as severe falls upon the head or back or any jarring concussion, but the exact relationship of the trauma to the development of the disease is obscure. Two cases I have met with were apparently due to repeated falls in the hunting field, another commenced within three days after severe jarring in a railway accident, another followed immediately after being run over by a motor car, and so on. In the large majority of cases of this disease, injury does not appear to be a feature, and syphilis can undoubtedly be excluded in spite of the efforts of certain writers to ascribe syphilis as the cause, on account of the resemblance to a weak syphilitic reaction in the Lange colloidal gold test of the cerebro-spinal fluid. It is mostly young women in the twenties who develop this curious disease, and no definite cause has yet been shown, although it has been seen to follow influenza, which sequence has been seen with locomotor ataxy and other chronic diseases.

In many respects disseminated sclerosis is the antithesis of tabes, young women instead of middle-aged men being affected, and stiffness with spasticity of the limbs being produced instead of hypotonia with loss of deep reflexes as in tabes. Disseminated sclerosis, unlike tabes dorsalis, rarely causes pains in the limbs or trunk, with the exception of pains accompanying flexor spasms of the legs in the late bed-ridden stages, but occasionally a girdle pain may be met with. One case had occipital neuralgia, another had gnawing pain in the bones; one commenced

with pain and stiffness in both legs, another had some pain with numbness of the inner fingers and forearm, called "neuritis," while a man complained of violent pains in the bladder with the desire for micturition, causing the legs to double up. His symptoms first began seven years before, after forcible retention of urine for six hours while at a party.

In a few cases I have seen neuritis of the trigeminal nerve present in the early stages of disseminated sclerosis. Such neuritis of the nerve trunk must be clearly distinguished from paroxysmal trigeminal neuralgia, another occasional complication.

A young woman *æt.* 32, with early disseminated sclerosis, had noticed two years previously numbness of the right half of the face and tongue, with loss of taste on the right half of the tongue, everything feeling cold in the mouth, though these symptoms all passed off in two months.

Another woman, *æt.* 41, had complained a year before I saw her of scalding pain in the right half of the tongue as far as the tip, the pain spreading into the chin. On examination she had well-marked anæsthesia over the whole right trigeminal area, with loss of taste on the right half of the tongue. Moreover, weakness of the left foot was ushered in by four days "sciatica," when ankle clonus, increased right knee jerk and extensor plantar reflex were found.

Oppenheim[1] refers to a patch of sclerosis at the emergence of the trigeminal nerve being associated with trigeminal neuralgia in disseminated sclerosis. In my own practice I have met with no fewer than seventeen cases of typical trigeminal neuralgia associated with chronic spastic paralysis, several of which were undoubtedly disseminated sclerosis, the others being chronic spastic paraplegia in older people in which the clinical picture of disseminated sclerosis was incomplete, though possibly in some of them the morbid condition might turn out to be identical (see p. 160).

[1] Oppenheim, "Textbook of Nervous Diseases." 5th ed. Trans. by Bruce, 1911. Vol. I., p. 337.

The fact remains that this combination of no fewer than seventeen cases of trigeminal neuralgia with spastic paralysis is too definite to be explained as a chance association.

Another remarkable point is that five of these seventeen cases were instances of bi-lateral trigeminal neuralgia, four of them characteristic advanced cases of disseminated sclerosis, while the fifth was a case of chronic spastic paraplegia in a woman with a history of syphilis, possibly also disseminated sclerosis. It would be natural to infer that the neuralgia in these cases of chronic cerebro-spinal disease must be central in origin, and due to sclerosis involving the nerve trunk or its fibres within the pons. Yet there is never any anæsthesia or other sign of lesion of the trigeminal nerve in these cases of paroxysmal neuralgia, and in every respect they behave identically with ordinary cases of chronic trigeminal neuralgia.

Alcohol injection of the nerve trunks at their foramina of exit at the skull instantly cures the pain in these cases in precisely the same manner as in ordinary cases of trigeminal neuralgia, which is not a result one would expect if the lesion preceding the neuralgia were central.

This association of spastic paralysis with trigeminal neuralgia which I have observed is therefore difficult to account for, but experience has shown me that it is equally amenable to alcohol injection treatment with the ordinary type, and the presence of the spastic paralysis need not be considered in the prognosis of the neuralgia (see pp. 158, 161).

CHAPTER XXII

VASOMOTOR DISORDERS

Diseases of the Blood Vessels

Chronic or paroxysmal pains, chiefly in the extremities, may be met with both in degenerative diseases of the blood vessels and in the spasmodic vasomotor disorders. Endarteritis and endophlebitis may be due to various causes, amongst which heredity is sometimes prominent; heavy and prolonged muscular work is a predisposing cause to arterial degeneration and in this country at least will affect the male sex mostly. Various toxic causes such as chronic alcoholism, excessive indulgence in tobacco and syphilitic disease are the common causes of chronic vascular degeneration, entailing loss of elasticity of the arteries with fibrous thickening of the middle coats and atheromatous changes and calcification in the inner coat. This thickening or hardening of the arteries, known as arterio-sclerosis, accompanied by high blood pressure, may also, it is thought, be produced by chronic supra-renal intoxication, and is said to have followed prolonged administration of supra-renal extract. Although the veins are unlikely to show the atheroma and calcification found in the arteries, yet the fibrous thickening found in the latter is also found in the smaller veins to a less degree, and even capillary sclerosis is said to occur. With this condition a vasomotor neuritis may develop and paroxysmal and somewhat less persistent pains may be very severe in the foot.

This condition often leads to gangrene, and may be met with in chronic diabetes in old people. The pain is of a burning and often stabbing character, and the skin of the feet is likely to be cold and bluish and excessively tender

in places, with slight swelling. Absence of pulsation in the dorsalis pedis and posterior tibial arteries may be noticed and in this condition the danger from gangrene is imminent. To avoid this and to mitigate the pain, the limb may be kept supported and the patient not allowed to put his foot to the ground for many weeks or even months until improvement occurs. The limb must be kept very warmly wrapped up and hot-water bottles used with care, as any burn will be excessively dangerous, and moist heat such as fomentations must be avoided, the skin of the foot and toes being dusted daily with starch and boracic powder. Analgesic drugs may be necessary to control the pain and to produce sleep, and combinations of aspirin, pyramidon and medinal, with the additions in the cases of intractable pain of heroin, omnopon, or morphine preparations. In suitable cases, insulin treatment will be advisable for the diabetes; dieting is very important, and simple food and very little alcohol are imperative, while a starch-free diet will be necessary in cases of diabetes. Sometimes, in spite of treatment, gangrene of the foot occurs and large portions of the foot may slough away, especially in cases of chronic tobacco poisoning.

Intermittent Claudication.—Chronic tobacco poisoning before it produces gangrene from arterio-sclerosis is liable to cause attacks of painful cramp owing to the insufficient blood supply to the limb due to commencing arterio-sclerosis.

This condition is commonest in members of the Hebrew race and usually affects the legs. No pain will be experienced while the sufferers from this complaint are at rest or moving about the house, but on going for a walk, even perhaps after fifty yards down the street, painful cramp in the calves will be set up and the patient must stop and rest for a few minutes before a further short progress can be undertaken. The condition is really an anæmic cramp of the muscles, which will function normally for slight movements, but when called upon for greater exertion, and therefore demanding a greater blood supply, which cannot be afforded

them on account of the inelastic condition of the arteries, painful cramp develops, which is really similar in type to the muscular cramps in the legs which occur in anæmic women when in bed, and is also comparable to angina pectoris, which is a relatively anæmic cramp of the heart muscle.

In some subjects these painful cramps of intermittent claudication may be met with in other muscles besides those of the lower extremities, and may occur in almost any muscle in the body which is used for prolonged exertion.

Angina cruris is a term sometimes applied to these painful cramps in the legs, and the same condition precisely may be seen after embolism of the popliteal artery.

Tobacco angina resembles angina pectoris due to coronary sclerosis in the painful cramps of the muscle, but it is less serious in the prognosis as a rule; usually tobacco angina, though it produces pain in the chest radiating into the neck and arm, is accompanied by hysterical phenomena, dyspnœa and excitement, the attack being much more prolonged than in the ordinary sclerotic angina. The source of the tobacco poisoning is not always obvious at first, as the patient may be a non-smoker. One very marked case of this complaint I saw in a clerk who used to take snuff regularly at a city divan while playing dominoes.

Functional Vasomotor Disorders

Various abnormal sensations ranging from itching, tingling, pricking and pins and needles sensations up to severe pain may be met with in functional vasomotor neuroses, including chilblain, dead fingers, Raynaud's disease, erythromelalgia, and acroparæsthesia. Chilblain—erythema pernio—a vasomotor disorder associated with chill, especially a damp cold, is often hereditary and is apt to be met with in persons of lymphatic temperament, with low blood pressure and slight anæmia. Pain is not a feature of this condition, though the itching that accompanies

the swelling of the fingers or toes, ears or any extremity, may be troublesome.

The deficient nutrition of the parts may lead to shallow ulcers being formed, and the defective growth of the nails during this period is very obvious later when the newer nail tissue has grown forward.

Dead fingers is a paroxysmal claudication of the arteries in the fingers or toes likely to be met with in cold weather in certain people and is sometimes considered to be a slighter degree of Raynaud's disease; occasionally it has been said to be associated with paroxysmal hæmoglobinuria.

With the attacks in which the fingers look cold, anæmic, and corpse-like, a certain amount of pain is liable to occur, while in the somewhat allied condition of **frostbite**, though no pain is experienced during the stage of the frozen tissue, during the thawing process while circulation is being restored pain may be intense. This stage of painlessness applies only to small areas of frostbite such as the tip of the nose, ears or fingers, but when larger areas of the body suffer from frostbite, as has been seen when men have been marooned upon an ice-floe, the agony experienced as frostbite seizes their limbs has been said to be almost indescribable.

Raynaud's Disease closely resembles in many ways chilblains and frostbite, commencing as local syncope in the extremities. This stage is succeeded by local asphyxia, or cyanosis, and lastly by gangrene. No pain is present in the stage of local syncope, but pricking, burning and even acute pain are liable to occur with the increasing depth of cyanosis and commencing gangrene. This disease is twice as common in women as in men and is very symmetrical. Attacks occur at first in the cold weather only, and with succeeding years the local asphyxia and cyanosis tend to become persistent, blebs form and ulcers develop, causing loss of tissue along the edges of the ears and finger tips, but only rarely does greater loss of tissue occur involving digits or even the loss of a hand or foot.

The condition is to be distinguished from chilblains or

The treatment of the acute abdominal attack will necessitate the use of opium internally, a drug which is naturally anathema to the surgeon in an acute abdomen. Other drugs, such as calcium and turpentine, have been used with good results.

Acroparæsthesiæ

The term signifies abnormal sensations at the extremities and is derived from the Greek *ἄκρον*, an extremity. This is not a clear-cut, definite disease, but the term includes a variety of abnormal sensations, some of which may be the prelude of an advancing spinal degeneration or a slight degree of multiple neuritis, the commencing stage of endarteritis obliterans, cervical or first rib pressure, anæmia, chronic rheumatism, goutiness, chronic indigestion and the menopause. From this list of unrelated conditions it is obvious that the symptoms included by different observers under the term acroparæsthesia may vary a good deal in quality and in prognostic importance.

Generally speaking, the term indicates numbness, deadness, pins and needles, pricking pains referred to the ends of the fingers and sometimes to the toes, and there may be slight changes in the colour of the extremities, which may be mottled, pallid or slightly cyanosed, and the definition is therefore liable to include all those cases of abnormal sensations and sensibility of the extremities which cannot be definitely labelled as peripheral neuritis, endarteritis, one of the vasomotor tropho-neuroses, etc., in which the symptoms do not develop beyond the stage of abnormal sensations and which does not lead to gangrene, erythromelalgia, or other definitely recognisable disease. The majority of cases are to be met with in middle-aged women about the time of the menopause, more particularly if somewhat anæmic, or suffering from chronic indigestion, or chronic rheumatism. The latter condition will indeed often be obvious from the presence of nodes upon the fingers, grating in the shoulder joints, a history of trouble-

some lumbago or other forms of muscular rheumatism, or neuro-fibrositis. Careful examination is, of course, necessary to exclude multiple neuritis, or chronic spinal cord disease. Glycosuria should be examined for, and amongst nervous diseases besides multiple neuritis combined sclerosis is perhaps the most likely to lead to error. More particularly should chronic spinal disease be suspected when the symptoms occur in a man over middle life. The reflexes must be carefully tested and any increase of knee jerk or doubtful extensor plantar reflex carefully noted, as the presence of such physical signs should exclude the diagnosis of acroparæsthesia and watch must be kept for further development of spinal symptoms.

At this first examination, for example, a man of 60 may show no increase of knee jerk, the plantar reflexes may be negative in character, and no complaint may be made of powers of walking or of balance, and the control of the sphincters may be perfect. Yet complaint of numbness and deadness of the extremities in a man of that age should make one very suspicious of the early development of multiple neuritis, of combined sclerosis, or of tabes dorsalis. The plantar reflexes should be examined from time to time and if the feet are cold at the time of the examination they should be placed for a few minutes in hot water, as often it will be found in the early development of spastic paralysis that plantar reflexes which cannot be elicited at all when the feet are cold will after warming the feet in hot water show a brisk extensor reflex movement. The recognition of tabes dorsalis will depend on a careful comparison of the deep reflexes on the two sides, the activity of the pupil reflexes, the presence of "rheumatic" shooting pains, ataxy, patchy anæsthesia, diplopia, sphincter weakness and so forth. If the deep reflexes are all equal and normal, both pupils equal and reacting well to light, and there are no shooting pains, the diagnosis of tabes is most unlikely. Sometimes disseminated sclerosis may commence with numbness of the extremities, though in this case there will usually be weakness and clumsiness of certain move-

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ments, probably increase of the deep reflexes, and an extensor plantar reflex.

The treatment of acroparæsthesia when organic diseases can be excluded is mainly symptomatic. Massage and electric baths, with tonics such as strychnine, arsenic, and iron will be the main line of defence, and sometimes the change to a different soil or climate may be followed by disappearance of the symptoms.

CHAPTER XXIII

NEURO-FIBROSITIS

Neuro-Fibrositis : Muscular Rheumatism

Chronic pains around the shoulder girdle, back of neck, lumbo-sacral region and around the hips are frequent in both sexes after the age of 30 to 35. Occurring as sudden stabs, usually local and not darting along a nerve trunk, but liable to be evoked by certain movements such as raising the arms to the head, turning the head, twisting the spine, stooping, etc., these pains are often loosely described as "muscular rheumatism" or as "neuritis." Their exact pathology is perhaps somewhat speculative, but there can be little doubt that the underlying morbid process is a chronic inflammation of the fibrous tissue of the great extensor muscular mass, which spreads from the sacrum along the back to the neck and back of the head. This chronic inflammation of the fibrous aponeuroses and intra-muscular septa may undoubtedly at times be truly rheumatic and allied to that acute infective process, rheumatic fever. Frequently cases of such chronic muscular rheumatism occur in the subjects of rheumatic fever, or may follow direct exposure to cold and damp. Many other similar cases, however, appear to arise as the result of chronic infection with organisms derived from various sources. Important amongst these is oral infection due to pyorrhœa, caries of the teeth and apical abscess. Other sources of infection are septic tonsillar crypts, chronic intestinal sepsis and appendicitis, though streptococcal infection from any septic focus may give rise to the condition. Well known, of course, as a cause of chronic fibrositis and joint disease is gonococcal rheumatism. The term fibrositis has been used for some years as a more

precise definition of the complaint than muscular rheumatism, and owing to the frequency with which nerve endings are involved in the scar resulting from this fibrositis, and the liability of the disease to spread to larger nerve trunks such as the sciatic, or the brachial plexus, I have suggested the term "neuro-fibrositis." A prominent symptom is tenderness on pressure over the muscles of the shoulder or back. This tenderness may be diffuse over the supra-scapular region and back of the neck, or along the side of the vertebral column just above the crest of the ilium, and above and internal to the great trochanter. When the tenderness is diffuse, without any special points of intense sensitiveness to pressure, treatment by alcohol injection or by the electrolytic needle is of no value, but often remarkable improvement can be obtained by a deep injection into the tissues of 10 to 15 cubic centimetres of 1 per cent. urea and quinine hydrochloride solution. This, of course, must be sterile, and it can be obtained in sterile ampoules of 5 and 10 cubic centimetres.

I have thus successfully treated many cases of chronic lumbo-sacral fibrositis in which pain and tenderness of the lower back and sacrum were a cause of chronic stiffness, with increase of the pain on movement. When this fibrositis shows signs of having spread towards the neighbourhood of the sciatic notch, by the presence of tenderness on pressure over the notch and the appearance of pains around the hip and down the back of the thigh, I find it a good practice to inject the sciatic notch itself with about 50 cubic centimetres of sterile saline. It is not actually necessary to inject the sciatic nerve itself with the saline, but this should be placed as near the exit of the nerve from the notch as possible. It would be dangerous to use alcohol here or even the urea and quinine hydrochloride, on account of the risk of damaging the sciatic nerve, but there need be no fear of damaging it when using the saline or saline and novocain, even if the nerve itself is thoroughly injected with the saline solution.

When it is found on palpation of the painful areas

that certain spots are acutely sensitive, the pressure of a finger or blunt pencil causing pain as though a sharp needle had been used, then a different treatment by alcohol injection may give most brilliant and immediate results. The tender spots must be very carefully localised and marked by a skin writing pencil, the commonest region for their occurrence being around the scapula, especially along its upper and inner borders. After sterilisation of the skin this should be anæsthetised by a vertical puncture with a hypodermic needle and 2 per cent. novocain. In many subjects the hypodermic needle, number 15 size, will be sufficiently long to reach the rib, which should be taken as a guide for the depth to which the needle may be sunk, as it is important that no alcohol injection should be made through the intercostal space into the pleura. If the tender point which has been localised is exactly between two ribs and over the intercostal space, the needle must be carefully pushed slightly upwards or downwards until the rib is felt, so that the proper depth is thus found and the needle may be then plunged vertically over the intercostal space into the tender point that has been marked, to the precise depth indicated by the rib. A drop or two of novocain should be injected as the needle puncture is made, and again when it reaches the proper depth. After waiting half a minute the syringe is changed for one containing 90 per cent. alcohol, and 7 to 10 minims of this are then injected, 3 or 4 more being injected as the needle is withdrawn. Often the patient complains at once of burning pains of a radiating character, perhaps as far as the shoulder and into the neck and back of head, and two or three spots may be injected at one sitting.

A farmer, a powerfully-built muscular man, some ten years previously slipped and fell when carrying a sack of wheat weighing about 260 lbs.

He was immediately seized with acute pain across the lumbar region, more especially in the neighbourhood of the right posterior iliac spine. Ever since he was subject to this pain, off and on, worse on movement or heavy

lifting, and occasionally to such severe exacerbations that he could scarcely move or dress himself. During an attack his spine was flexed laterally towards the right, and there was a small circumscribed area of great tenderness on deep pressure over the right posterior iliac spine. The spot was carefully localised and injected deeply into the tender area with a few minims of 90 per cent. alcohol. This completely relieved his pain within fifteen or twenty seconds, so that he was able to stand up straight, the lateral flexion of the spine having disappeared, and he walked away in comfort. Three years later he wrote that he was still well and able to do full work.

A lady, *æt.* 60, had for eight years been treated for pain in the right upper arm, worst in the front of the arm just below the deltoid, and also behind the shoulder. She had been treated in numerous spas by a large variety of methods, but the pain had become steadily worse, so that when I saw her she was never free from pain for five minutes, and was unable to lie on her right side. Sewing or writing would bring on severe pain in the wrist or fingers, but all movements of the arm were perfectly free, and there was no evidence of any arthritis. Examination disclosed the presence of definite tender spots on pressure under the pectoral tendon in the front of the arm, and also in the back slightly to the right side of the 7th dorsal spine, and in the upper right forearm on its posterior aspect, two inches below the elbow. All three of these tender spots were injected deeply with strong alcohol, ten to fifteen minims each. Three days later she stated that there was no pain at all in the arm, and five weeks later her doctor wrote that she was well and had practically no discomfort.

In Jan. 1922 I saw a man *æt.* 29, who complained of pain in his back with every muscular effort since his schooldays, when *æt.* 17 he ricked his back at football. On examination I found a tender spot on each side of the erector spinæ, three inches from the middle line and rather less above the nearest point of the crest of the ilium. The two tender points were closely symmetrical, and pressure on them inwards and forwards caused acute pain. These spots were then injected deeply with strong alcohol, the needle being inserted to a depth of $1\frac{1}{2}$ inches. The result was excellent and immediate. A year later he wrote

that he was quite well, no pain whatever, and that he had been able to dig drains and to work with pick and shovel in the building of a farm.

Similar immediate and dramatic cure may be sometimes wrought in cases of long-existing tenderness and pain in the feet from chronic rheumatism and metatarsalgia. Search may disclose two or more acutely tender spots on pressure on the sole, and these should be punctured down to the bone with a fine needle, and injected with a few drops of strong alcohol.

The effect of the alcohol is to destroy any nerve fibrils that it reaches, and the persistently sensitive spots that have been previously located probably indicate the site of involvement of a nerve fibril in the fibrositic scar tissue. The alcohol causes rather sharp burning pain for about fifteen or twenty seconds, after which the part feels fairly comfortable for an hour or more. Considerable aching pain may then be felt for the first night, which should, if necessary, be relieved by pyramidon, medinal and heroin cachets, or by a hypodermic injection of morphine. By next day the pain will be gone and the patient feels the benefit of the disappearance of the tender spots, as now the arm or back can be moved with impunity and the whole trouble may thus be cleared up by one injection. It is indeed remarkable for how long such chronic localised tender areas may persist, and I have thus relieved cases where the local pain has persisted for over seven years. Another method of treating these tender spots by electrolysis has been practised by Dr. Naunton Davies, who uses a long, sharp needle insulated by shellac except for the point. This needle is then plunged into the tender point and its other end connected through a needle holder and wire to the negative pole of a galvanic battery.

Acupuncture was recommended by Ringer for the treatment of these conditions and for chronic sciatica forty years ago, half a dozen or more needles being plunged into the tender areas or along the course of the sciatic nerve. Good results have often been obtained by this

method, but when one or more local spots of intense tenderness are discovered, I consider that alcohol puncture is far more valuable.

Subsequent treatment by diathermy, massage and Droitwich deep saline baths is a useful completion to the treatment, and may help to prolong the cure, though often the alcohol piqure alone has proved quite satisfactory.

Sacro-iliac Sprain or Subluxation.—Chronic pain around the buttock and lower part of the back with tenderness on pressure over the neighbourhood of the posterior superior iliac spine is very commonly treated as lumbo-sacral fibrositis or sciatica without benefit, perhaps for years, the patient complaining of pain on walking or twisting the back, while often, especially in the female, signs of neurosis and tenderness of the vertebral spines appear. Dramatic cures of this condition have been performed by osteopaths and cheiropractors, and orthopædic surgeons are now familiar with the condition, which is known in America as subluxation of the sacro-iliac joint. X-rays, however, have failed to demonstrate any evidence of displacement or disease in this joint, yet manual treatment and wrenching may cure the condition at once. A sudden audible click is often heard by those standing near, the noise having been quoted as evidence of the replacement of the subluxation of the joint. This treatment should be done under an anæsthetic, such as gas and ether, in order to obtain perfect results, when the joint is wrenched by a sideways twisting movement of the pelvis, or else the leg is strongly flexed up on the abdomen with the knee kept straight, thus over-stretching the ham-string muscles.

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